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EARLY TENSILE PROPERTIES OF HEALING CHICKEN FLEXOR TENDONS: A COMPARISON OF POSTOPERATIVE IMMOBILIZATION AND EARLY CONTROLLED PASSIVE MOTION

by

LYNNE M. FEEHAN

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH

IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE

OF MASTER OF SCIENCE

DEPARTMENT OF PHYSICAL THERAPY

EDMONTON, ALBERTA

SPRING, 1988

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ISBN 0-315-42827-9

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DEGREE: MASTER OF SCIENCE

YEAR DEGREE GRANTED: SPRING, 1988

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled EARLY TENSILE PROPERTIES OF HEALING CHICKEN FLEXOR TENDONS: A COMPARISON OF POSTOPERATIVE IMMOBILIZATION AND EARLY CONTROLLED PASSIVE MOTION submitted by LYNNE M. FEEHAN in partial fulfillment of the requirements for the degree of MASTER OF SCIENCE.

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ABSTRACT

Tensile properties of healing flexor tendons were examined in 30 chickens during the first 30 days after a surgical division and repair of the profundus tendon in the middle toe of each foot. Commencing on the 4th postoperative day, one foot was managed by a twice daily, five-minute session of controlled passive motion. The contralateral foot remained immobilized and acted as a matched control.

Six chickens were then sacrificed at each of 5, randomly assigned intervals (10, 15, 20, 25 and 30 days). The repaired tendons were then dissected out and immediately deep frozen until tensile testing. Prior to tensile testing all tendons were thawed in saline and measured for cross-sectional area. All testing was conducted on a specially adapted Instron testing apparatus, at a strain rate of 2.54 cm/min, and under standardized environmental conditions (37^{o}C, >98% RH).

Results of the 2 x 5 (treatment condition by healing period) analysis of variance showed: (1) significantly greater values for the treatment group for load, stress and energy absorbed to rupture, $p \leq .05$; (2) a significant increase in material stiffness and stress, and a significant decrease in strain, for both treatment groups across the 20-day healing period examined, $p \leq .05$; and (3) no significant difference between the groups in rate of change for any of the tensile properties examined, $p \leq .05$.

Findings from this study support the role of controlled passive motion in improving the structural and material tensile properties of healing chicken flexor tendons during the initial thirty postoperative days. Clinically this suggests, that the use of early controlled passive motion following primary tendon repair should similarly improve the early functional tensile properties in healing human flexor tendons.

ACKNOWLEDGEMENTS

I would like to begin by thanking my committee members, Dr S. Kumar, Dr D. Ford, Dr J. G. Beauchene and Dr M. Wayman, for their participation in this project.

I would especially like to acknowledge the contributions of Dr's Ford and Beauchene. Dr Ford for her ongoing support and encouragement, and Dr Beauchene for his unbelievable generosity in giving freely his time, surgical expertise and financial assistance. Without Dr Beauchene's assistance this project would not have been completed.

There are also two other people who's contributions were integral to the completion of this study. Trudy Hoogan, from the Surgical Medical Research Institute (SMRI), and Bob Konzuk, from the Department of Mineral, Mining, Metallurgical and Petroleum Engineering. Trudy coordinated and assisted in the surgical procedures, and managed to get things to run smoothly with the minimal amount of fuss. Bob did so many things related to the design and completion of the tensile testing procedures that it is impossible to list them all. Without Bob's assistance, and the cooperation of his department, this study again would not have been completed.

I would also like to thank the animal care technicians from the SMRI. Appropriate care of animals in a study of this nature is so very important.

The Department of Anatomy and Cell Biology was also kind enough to allow me to use their ultralow freezer, and allow their department photogragher, Greg Morrison, to assist with my photography.

Finally, and most importantly, I would like to thank Greg Feehan. Greg was always there, providing me with the love and support I needed. Thank you Greg, ILYKK.

* This study was funded in part from the Celia Bartzen Graduate Bursary, provided by the Edmonton District of the Canadian Physiotherapy Association.

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Chapter I

INTRODUCTION

Background to the problem

Tethering of a primarily repaired flexor tendon to the surrounding fibro-osseous tunnel in the finger presents a significant clinical problem. Adherence of the tendon limits the tendon's ability to glide, which results in decreased finger active range of motion following primary surgical repair. With maximal functional recovery being a defined rehabilitation goal, establishing an effective means for overcoming the restrictive influence of this adherent scar is of considerable importance.

Surgical management of flexor tendon injuries in the fibro-osseous tunnel has been a subject of discussion in the medical literature for some time. Bunnell (1918), described this region as "no-mans-land" and advocated secondary tendon grafting procedures for injuries in this region. In the past twenty years, however, the surgical trend has shifted away from secondary tendon grafting towards primary tendon repair of acute flexor tendon injuries in the fibro-osseous tunnel (Verdan, 1960 & 1972).

Kleinert and Verdan (1983), reported on a study involving an unspecified number of Scandinavian patients. They describe 63% good to excellent results with primary tendon repairs as compared to 35% good to excellent results with

tendon grafting in the fibro-osseous tunnel. Other clinical studies have reported comparable good to excellent results following primary tendon repair (Green & Niebauer, 1974; Kleinert, Kutz & Cohen, 1975; Strickland & Glogovoc, 1980). Although this trend towards primary repair of flexor tendons in the fibro-osseous tunnel seems to be supported by these reported clinical results, the overall functional results are still less than optimal.

Many factors have been described as affecting the ultimate functional results of primary tendon repairs in this region. Some of these factors are; skill and technique of the surgeon (Verdan, 1972), age of the patient (Amadio & Hunter, 1987), method and type of suture (Kessler & Nissan, 1969; Urbaniak, Cahill & Mortensen, 1975; Ketchum, Mortin & Kappel, 1977; Wray & Weeks, 1980), integrity of the blood vascular system or vincula (Strickland, 1985), integrity of the flexor sheath (Ketchum, 1977; Kleinert, Schepels & Gill, 1981; Strickland, 1983 & 1985; Lister, 1985) and the early postoperative management regimes (Strickland & Glogovoc, 1980; Kleinert & Verdan, 1983).

Clinical hand therapists have no control over such factors as age of the patient, site and type of injury, extent of damage to the vincula and sheath, and the skill and surgical technique of the hand surgeon. However, the therapist should be cognizant of these factors as they affect the postoperative management regimes and the ultimate functional

result. Conversely, early postoperative management of primarily repaired flexor tendon injuries is the responsibility of hand therapists and evaluation of the effectiveness and validity of the various early postoperative regimes should be critically evaluated by them.

There are three different approaches to the early postoperative management of primaraly repaired flexor tendons described in the literature. These are:

1. Early postoperative immobilization.
2. Early postoperative active mobilization.
3. Early postoperative controlled passive mobilization.

Early postoperative immobilization has been the traditional management advocated following primarily repair of flexor tendons in the fibro-osseous tunnel. This regime consists of three weeks of immobilization, followed by graduated active tendon mobilization over the next several weeks (Mason & Allen, 1941; Peacock, Madden & Trier, 1971). Flexor tendon injuries managed in this fashion heal with considerable scar and demonstrate a significant loss in active range of motion in the affected digit (Strickland & Glogovoc, 1980).

Other studies have advocated the use of early postoperative active tendon mobilization (Lahey, 1923; Hernandez, Velasce, Rivas & Preciado, 1967; Kessler & Nissam, 1969; Becker, Orak & Duponsell, 1979; Hester, Hill & Nahai,

1985; MacMillan, Sheppard & Dell, 1987). However, because of such factors as complicated surgical approaches, high incidence of tendon rupture and bulky repair sites these approaches have failed to develop wide clinical acceptance.

The use of early postoperative controlled passive motion has also been advocated (Duran & Houser, 1975; Lister, Kleinert, Kutz & Alasoy, 1977). Clinical studies showing improved digital function with early controlled passive motion (Strickland & Glogovoc, 1980; Kleinert & Verdan, 1983) have helped to establish clinical acceptance of this postoperative management regime. Initial experimental studies, conducted primarily on dog and chicken animal models, have also shown some interesting results. In addition to improved tendon excursion (Young, Weeks & Wray, 1981), tendons managed in this fashion have shown evidence of healing through an intrinsic healing process without scar formation (Gelberman, Vandeberg, Manske & Akeson, 1985; Nelson, Heiple, Shaffer, Keith, Lacey, Davy & Paxirandeh, 1985) and have demonstrated greater rupture strength throughout the third to twelfth week (Gelberman, Woo, Lothringer, Akeson & Amiel, 1982) and first to fifth week (Hitchcock, Light, Bunch, Knight, Sartori, Patwardhan & Hollyfield, 1987) of postoperative healing.

Two factors have been presented in the literature that may contribute to these beneficial experimental effects of early controlled passive motion on healing flexor tendons. These

factors are; the role of dynamic synovial fluid nutritional pathways in healing flexor tendons (McDowell & Synder, 1977, Weber, 1979 & 1987), and the effect of applied tensile stress on the strength of healing connective tissues (Mason & Allen, 1941, Thorngate & Ferguson, 1958, Becker & Diegelmann, 1984).

The first factor, the importance of motion for synovial fluid diffusion within the fibro-osseous tunnel was first proposed by McDowell & Schneider (1977). Tendon motion has since been shown experimentally (Weber, 1979 & 1987) to facilitate the diffusion of fluorescein marked synovial fluid into the central portion of flexor tendons from the hypovascular regions of the tendon. This concept of motion being an integral element for flexor tendon nutrition in the fibro-osseous tunnel is important when considering the increased nutritional requirements of a healing tendon.

Injury and surgical repair can affect healing potential by interfering with the flexor tendon's delicate vascular integrity within the fibro-osseous tunnel (Matsui, Jaeger, Merklin & Hunter, 1987). Postoperative immobilization of healing flexor tendons can further compromise healing by diminishing the dynamic synovial fluid diffusion pathway within the tendon. Whereas, early controlled passive motion regimes following primary tendon repair in the fibro-osseous tunnel may enhance early tendon healing by improving this dynamic tendon synovial fluid diffusion nutritional pathway.

The second factor, the beneficial effect of early tensile

stress on the strength of healing flexor tendons was first reported by Mason and Allen (1941). These authors noted that active motion initiated at three weeks postoperatively, significantly improved the rupture strength of healing tendons when compared to immobilized tendons. Thorngate and Ferguson (1958), examined the effect of stress on healing aponeurotic wounds in rabbits and concluded that aponeurotic wounds which heal under tension have a greater bursting strength than wounds healing without tension. Gelberman et al (1982), examined the tensile rupture strength of healing flexor tendons managed by delayed motion, immobilization, and early passive motion regimes and found that the early mobilized groups had twice the rupture strength throughout the third to twelfth postoperative week healing periods than did the immobilized tendon group. Hitchcock et al (1987), examined the effect of early constrained digital active and passive motion on the rupture strength of healing flexor tendons in chickens and found significantly greater rupture strength for flexor tendons managed by early motion throughout the 5th to 40th day healing period as compared to tendons managed with postoperative immobilization.

In a review article, Bassett (1971), described four possible mechanisms for beneficial effects of stress on connective tissues. The possible mechanisms are:

1. That the micro-environmental conditions of the tissue, such as oxygen tension and physical forces,

direct the pathway of specialization of undifferentiated mesenchymal cells into bone, cartilage or fibrous connective tissue cells.

2. That applied physical forces control macromolecular orientation by generating piezoelectric forces within the connective tissue.
3. That stress-induced potential differences on the surface of collagen fibers may also control collagen fiber bundle aggregation and maturation.
4. That motion-induced electrical streaming potentials in tendon may massage body fluid through the extracellular matrix.

Bassett (1971), felt that these four factors were evidence that both nutritional factors and physical forces determined in part the direction of cell specialization and behavior within connective tissues.

The previously described findings supportive of both the importance of motion for synovial fluid diffusion within tendon and the beneficial effect of stress on healing tendons, suggest that a controlled passive motion regime during the early postoperative healing period has the potential to play a role in:

1. Directing the nature and rate of early healing processes.
2. Enhancing the development of morphological changes

within the healing tendon that may contribute to an enhanced development of its functional tensile properties.

Statement of the problem

The question of when to commence active tendon mobilization following primary flexor tendon repairs that have been managed by an early postoperative controlled passive motion regimes has not yet been addressed fully in the literature. Traditionally, active tendon mobilization is usually commenced at three weeks following an early postoperative immobilization regime for primary flexor tendon repair in the fibro-osseous tunnel (Mason & Allen, 1941; Peacock et al, 1971). In contrast, when early controlled passive motion regimes are utilized, initiation of active motion has not been advocated until four and one half weeks (Cannon & Strickland, 1985), five and one half weeks (Duran, Houser, Coleman & Stover, 1987), and even up to ten weeks (Kutz, 1987) postoperatively.

The reasons for this delay in active tendon motion with early controlled passive motion are unclear. Caution, for fear of rupture, has perhaps been advocated on the assumption that tendons managed by controlled passive motion heal more slowly, and so develop tensile strength more slowly than tendons managed by early postoperative immobilization.

The previously mentioned findings for the importance of

motion for synovial fluid diffusion within tendon, and the beneficial effect of stress on healing connective tissues, suggests that the opposite may be true. Tendons managed with controlled passive motion regimes during the early postoperative healing period have the potential to:

1. Heal with a more efficient early healing process due to improved synovial fluid nutrition.
2. Develop morphological changes that facilitate development of early functional tensile material properties due to the beneficial effects of stress on healing connective tissues.

Unfortunately, no studies have been reported that examine differential effects of early postoperative management regimes on the early material tensile properties of healing flexor tendons.

Objectives of the study

The primary objective of this study was to compare the effect of early postoperative controlled passive motion and early postoperative immobilization regimes on the material tensile properties of healing chicken flexor tendons during the initial thirty day postoperative period. More specifically, this study was conducted in a controlled experimental setting, to examine the early material tensile properties (load at initial failure, stress at initial

failure, strain to initial failure, material stiffness and energy absorbed to initial failure) of surgically divided and repaired chicken flexor tendons at five specific postoperative intervals (10, 15, 20, 25 and 30 days), and to compare these tensile properties between tendons managed by early postoperative controlled passive motion and tendons managed by early postoperative immobilization.

Significance of the study

An investigation of specific material tensile properties of healing flexor tendons during the early healing period can provide important information regarding the development of functional tensile properties of the healing tendon. A comparison of these functional tensile properties between tendons managed with either postoperative immobilization or early controlled passive motion would also provide important insights into potential differences in type, rate and efficiency of early healing processes of healing flexor tendons.

With this knowledge of early material tensile properties following two different early postoperative management regimes, clinicians can better judge both the propriety of these regimes and the timing of postoperative active tendon mobilization programs.

Research theory and null hypotheses

The reported findings for the importance of motion for synovial fluid diffusion within tendons and the beneficial effect of stress on healing tendons, suggest that controlled passive motion regimes during the early postoperative healing period would have the potential to play a role in:

1. Directing the nature and rate of early healing processes.
2. Facilitating the development of morphological changes within the healing tendons that contribute to the development of functional tensile properties of the healing flexor tendon.

Based on these reported findings of beneficial effects of early controlled passive motion on flexor tendon healing, a comparison of early postoperative material tensile properties of healing flexor tendons managed by either early postoperative immobilization or early postoperative controlled passive motion, should reflect potential differences in type, rate and efficiency of early healing processes.

The specific research null hypotheses for an analysis of variance of the effects of treatment condition (early postoperative immobilization vs early postoperative controlled passive motion) and healing period (10, 15, 20, 25 and 30 days) on the material tensile properties of the

healing chicken flexor tendons were:

1. Treatment effect: That the early controlled passive motion tendon treatment group would not have significantly different values for any of the tensile properties examined when compared to the early postoperative immobilization tendon treatment group.
2. Healing effect: That tendons in both the early postoperative immobilization and the early controlled passive motion treatment groups would show no significant change across the five healing periods examined in any of the material tensile properties.
3. Interaction effect: That the early controlled passive motion tendon treatment group would not have a significantly different rate of change in any of the tensile properties examined as compared to the early postoperative immobilization treatment group.

Delimitations

There are several accepted clinical approaches to surgical repair of lacerated flexor tendons in the fibro-osseous tunnel in the human finger. This study utilized a strictly controlled surgical division and repair that replicated the clinically ideal primary surgical repair (Strickland, 1985) of an uncomplicated Flexor Digitorum Profundus tendon laceration at the level of the distal one quarter of the

proximal phalanx in the fibro-osseous tunnel of the human finger.

Tensile properties of tendons vary significantly with environmental testing conditions, strain rate, and the age/activity level/ and sex of the test specimen (Galante, 1967; Viidik, 1973; Tipton, Matthes, Maynard & Carey, 1975; Noyes & Grood, 1976). This study was conducted only on young mature white leghorn roosters of the same genetic breeding pool and similar age (27 - 31 weeks). All chickens were limited to cage activity only for one week prior to surgery. All tendons underwent a uniaxial tensile test under the same environmental conditions (37° C, >98% R. H.) and strain rate (2.54 cm/min; 100% specimen elongation/min).

Tensile properties of tendon will also change with temporal changes in material composition of the healing tendon. This study only examined the changes in tensile properties of healing chicken flexor tendons during the early 10 to 30 day postoperative healing period.

Limitations

In this study a chicken toe animal model was selected because of the anatomical similarities between chicken toe and human finger flexor mechanisms (Farkas, Thomson & Martin, 1974). Although the chicken toe anatomy has a similar intimate tendon/fibro-osseous synovial sheath arrangement and vascular supply as the human finger, it differs both in the

size and number of tendons (3 vs 2) found in the digit (Figures 1.1, 1.2 and 1.3).

Application of these experimental findings to other than the experimental setting may also be limited by possible physiological differences in the healing processes between chickens and humans. Unfortunately, a direct examination of the early tensile properties of healing human flexor tendon is not possible.

Operational definitions

Fibro-osseous tunnel: A specialized thickening of the deep volar connective tissue layer in the finger, originating from either side of the deep bony tissue and surrounding the flexor tendons. The fibro-osseous tunnel forms a close fitting specialized sheath through which the flexor tendons pass. The outer surface has a series of thickened connective tissue bands that act as pulleys and keep the tendons in close contact with the underlying bone at all times. The inner surface of the sheath, and the outer surface of the flexor tendons, are each covered by a layer of synovial membrane. The synovial fluid produced by this membrane acts both as an important source of nutrition for the tendons and as a lubricating fluid to allow friction free motion of the tendons within the sheath.

Early postoperative period: The initial three to four week period following surgery. This period corresponds with

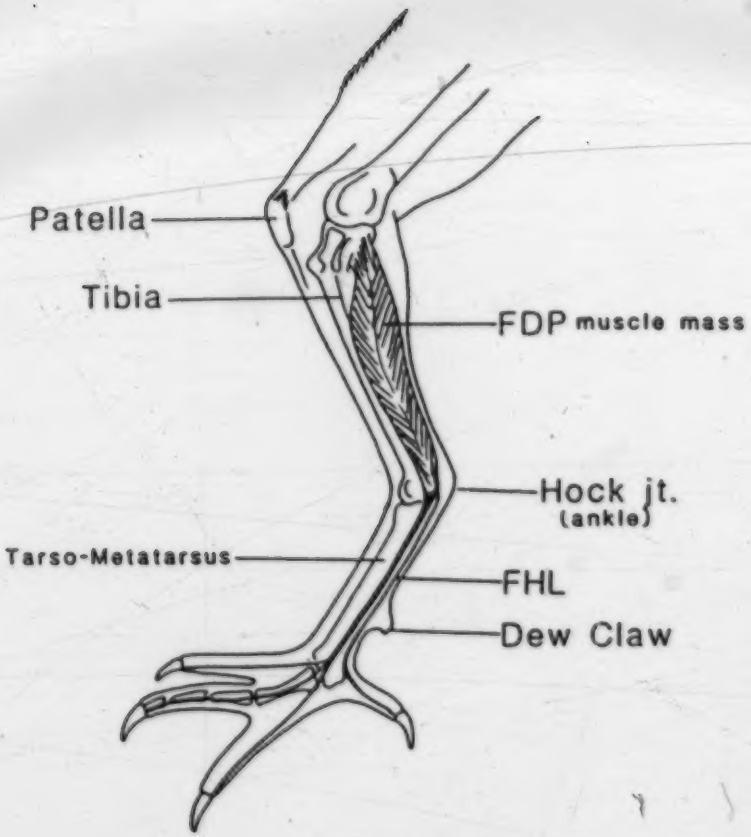

Figure 1.1 Chicken Leg - Osseous Anatomy and Flexor Digitorum Profundus Muscle.

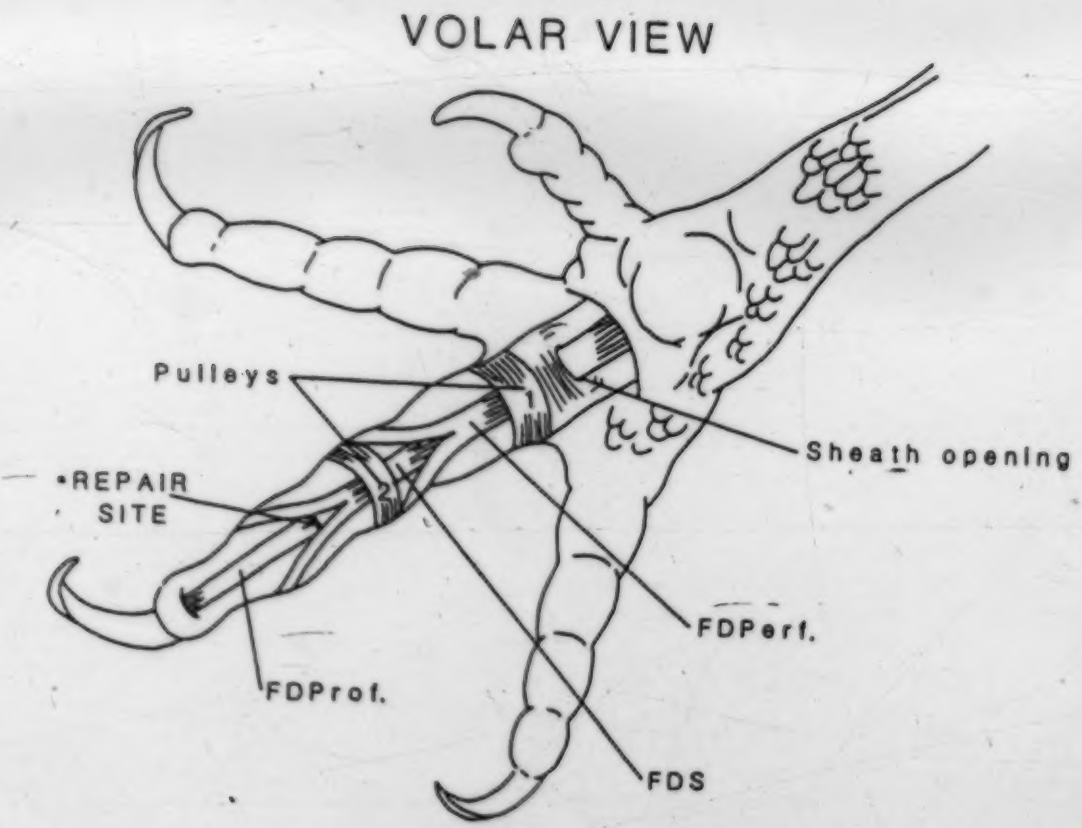

Figure 1.2: Chicken Digital Flexor Mechanism (Volar view)

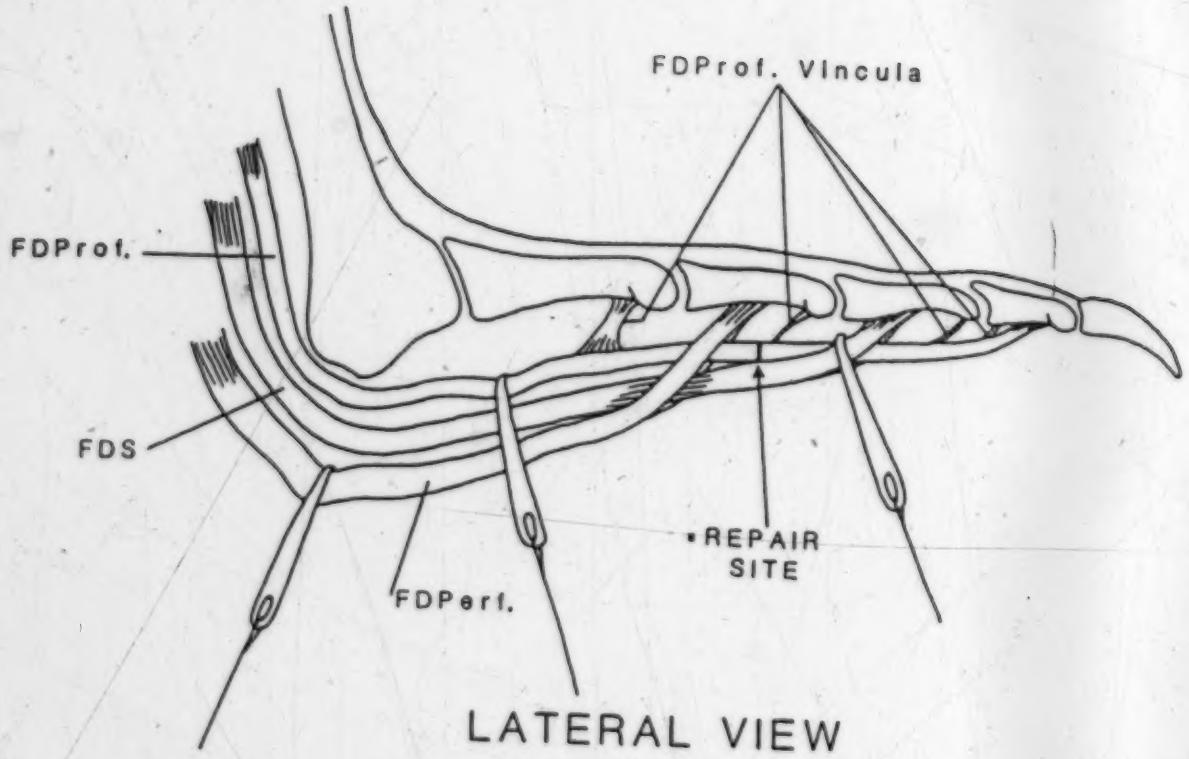

Figure 1.3: Chicken Digital Flexor Mechanism (Lateral view)

the inflammatory and fibroblastic phases of connective tissue healing.

Controlled passive mobilization: Movement of the flexor tendon through closely monitored passive joint range of motion exercises.

Active tendon mobilization: Movement of the flexor tendon through active muscle contraction exercises.

Tensile properties: The mechanical properties (eg. strength and stiffness) of the tendon, demonstrated through a uniaxial tensile test.

Load: The amount of tensile load (grams) applied to the tendon specimen at any given time during the tensile test.

Stress: The internal resistance of the tendon to an applied load (grams) per unit area (square millimeters).

Strain: The relative displacement of two points in the tendon per unit separation. Presented in this paper as percentage of total specimen length.

Stiffness: A measure of the rate of deformation of the tendon in relation to the tendon's rate of change in load bearing capabilities. Determined by dividing stress by strain.

Initial failure: The initial point on the load / elongation curve that demonstrated a loss in ability of the tendon to resist the applied load.

Energy absorbed to initial failure: A measure of the energy stored in the tendon. This is measured by determining

the area under the load / elongation curve from the point of initial strain up to the point of strain to initial failure. Energy absorbed is a function of both the stiffness and strength of the tendon.

Elastic strain: Deformation of the tendon that is completely and immediately recovered when the tensile load is removed.

Visco-elastic strain: Deformation of the tendon that is completely recovered over a period of time following removal of the tensile load.

Plastic strain: Deformation of the tendon that is permanently maintained following removal of the tensile load.

Chapter II

LITERATURE REVIEW

Surgical management

Effective surgical management of acute flexor tendon lacerations ~~in~~ the fibro-osseous tunnel of the finger has been a subject of discussion in the medical literature for some time. Predictably poor functional results following primary tendon repair of flexor tendons in the fibro-osseous tunnel led Bunnell (1918) to describe this region as "no-mans-land". Bunnell advocated secondary tendon grafting procedures for flexor tendon injuries in this region. He felt that better surgical conditions with secondary grafting procedures would result in less postoperative scarring and ultimately lead to better clinical results.

More recently, Verdan (1960 & 1972) has advocated the return to primary tendon repair of acute flexor tendon lacerations in what he described as zone II flexor tendon injuries. Verdan (1972), felt that results of primary flexor tendon repairs in Zone II were primarily dependent upon the meticulous surgical technique of "knowledgeable and skilled" surgeons.

Zone II flexor tendon injuries have since been more precisely defined (Kleinert, Kutz, Ashbell & Martinez, 1967; Strickland, 1983) as the portion of the fibro-osseous tunnel between the first annular pulley and the insertion of the

Flexor Digitorum Sublimus (FDS) tendon into the middle phalanx. Zone II is the region in the finger in which both flexor tendons run intimately within the restrictive fibro-osseous tunnel and adhesive scar formation with tendon healing has its most restrictive influence within this region.

Kleinert and Verdan (1983), reported on a Scandinavian series of patients (unspecified number) and described 63% good tc excellent results with primary tendon repairs as compared to 35% good to excellent results with tendon grafting in zone II of the fibro-osseous tunnel. Other clinical studies have reported comparable good to excellent results following primary flexor tendon repair in zone II (Green & Niebauer, 1974; Kleinert, Kutz & Cohen, 1975; Strickland & Glogovoc, 1980). Although this trend towards primary repair of flexor tendons in zone II injuries in the fibro-osseous tunnel seems to be supported by these improved clinical results, the ultimate functional recovery of the affected digits is still less than optimal.

Besides meticulous surgical technique (Verdan, 1972), other factors have been described as affecting the ultimate functional recovery of primary tendon repairs in zone II. Some of these other factors are; age of the patient (Amadio & Hunter, 1987), method and type of suture (Kessler & Nissan, 1969; Urbaniak, Cahill & Mortensen, 1975; Ketchum, Mortin & Kappel, 1977; Wray & Weeks, 1980), integrity of the blood

vascular system or vincula (Strickland, 1985), integrity of the flexor sheath (Ketchum, 1977; Kleinert et al, 1981; Strickland, 1983 & 1985; Lister, 1985) and, early postoperative management regimes (Strickland & Glogovoc, 1980; Kleinert & Verdan, 1983).

Strickland (1985) and Kleinert and Cash (1987), summarize important technical considerations of a primary surgical repair of a clean, uncomplicated laceration of a FDP tendon in zone II of the finger. Some factors these authors stress are the importance of surgical repair by a knowledgeable surgeon, use of loupe magnification, adequate surgical exposure and atraumatic technique. These authors also discuss the consideration of placing the suture in the avascular, volar portion of the tendon to avoid further damage to the vascular supply of the tendon.

A technique of tendon suturing that is widely accepted for the repair of flexor tendons in zone II is also described by Strickland (1985) (Figure 2.1). The suturing technique involves a Tajima (1984) modification of a Kessler & Nissan (1969) grasping suture, in which the knot is located at the site of laceration. The repair technique described by Strickland (1985) also includes his own modification, in which the suture knot is buried between the two ends of the tendon by a small 6-0 running epitenon suture.

Lister (1985), stresses the importance of tendon sheath closure in light of the reported experimental findings of the

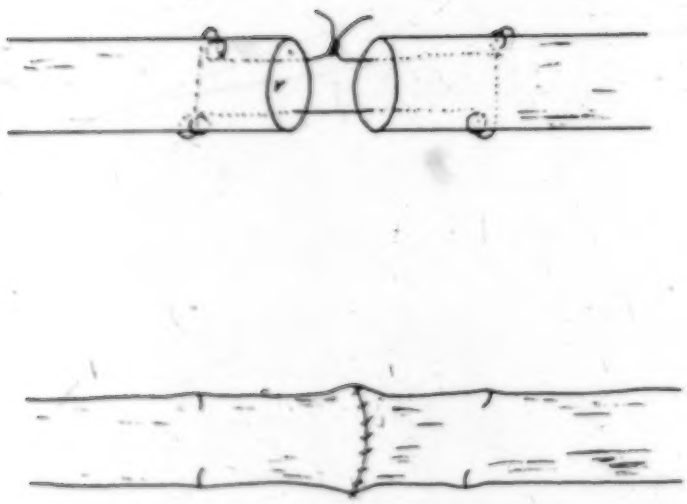

Figure 2.1: Flexor Tendon Suturing Technique. (Adapted from: Strickland, 1985).

importance of synovial fluid nutrition for flexor tendons in the fibro-osseous tunnel of the finger. He describes in detail the surgical techniques for ensuring preservation and repair of the flexor tendon sheath during a primary repair of a flexor tendon.

In summary, the surgical trend of the past 20 years away from secondary tendon grafting procedures, towards primary tendon repairs for acute flexor tendon lacerations in the fibro-osseous tunnel of the finger, appears to be supported by the reported clinical findings of improved functional recovery of the digit with primary tendon repair. However, the ultimate functional recovery of the affected digit is still less than optimal.

Many factors have been identified as affecting the ultimate functional recovery of the affected digit. Clinical hand therapists have no control over many of the factors described, such as age of the patient, site and type of injury, extent of damage to the vincula and sheath, and the skill and surgical technique of the hand surgeon. However, the therapist should be cognizant of these factors as they may affect postoperative management regimes and the ultimate functional result.

Early postoperative management

In contrast to the numerous factors that are outside the control of the hand therapist, management of the early

postoperative treatment regimes is the responsibility of the hand therapist. For this reason, evaluation as to the effectiveness and validity of the various approaches to early postoperative management of these injuries should also the therapist's responsibility. Many important clinical questions can not be addressed without further experimental and clinical research conducted to examine the effectiveness of the different early postoperative management regimes.

A universal, or widely accepted approach to the early postoperative management of primarily repaired flexor tendons in the fibro-osseous tunnel of the finger has not as yet been clearly established. Presently there are three approaches to the early postoperative management of these injuries described. These are:

1. Early postoperative immobilization.
2. Early postoperative active mobilization.
3. Early postoperative controlled passive mobilization.

The ultimate goal of postoperative management of a primarily repaired flexor tendon in the finger is to maximize digital functional recovery. Functional return of the digit is dependent upon more than just a well approximated and strongly healed tendon. In order for the digit to function, the healed tendon must also glide freely in the fibro-osseous tunnel of a mobile and supple finger.

Tethering of the healing tendon to the surrounding fibro-

osseous tunnel following primary tendon repair presents a significant clinical problem. Prolonged immobilization and adhesfve scar formation during the early stages of healing ultimately leads to reduced finger function. Therefore, establishment of an early postoperative management regime that both protects the tendon from excessive tensile forces and reduces the restrictive influence of adherent scar, becomes an important rehabilitation challenge.

Of the three different approaches to the early postoperative management following primary tendon repair in the fibro-osseous tunnel of the finger, early postoperative immobilization has been the traditional management regime advocated. The protocol for this regime has been described by Peacock (1964) and Peacock et al (1971), and is based on a "one wound -one scar" concept of flexor tendon healing in the finger. Peacock (1964) describes the injured flexor tendon as being part of a multiple tissue wound, in which the scar that eventually repairs this wound is, during the early stages of inflammation and fibroplasia, one mass of reparative tissue. Peacock et al (1971) state, that if this reparative scar is to develop vascularization and adequate tensile strength to withstand active motion, a period of three weeks of postoperative immobilization was required. They also state, that the process of secondary remodelling of the reparative scar into tendon like tissue centrally and nonrestrictive connective tissue peripherally, was dependent

upon the appropriate and cautious initiation of a graduated active tendon mobilization following the three week period of immobilization.

The postoperative management regime described by Peacock et al (1971), requires that the hand and wrist are immobilized in a bulky compressive dressing with the wrist positioned in neutral or slight flexion and the metacarpal phalangeal (MCP) and interphalangeal (IP) joints flexed to relieve tension across the repair site. The hand is left immobilized in this position for three weeks. Following removal of the dressing, a program of unresisted active blocked proximal IP joint motion, and passive digital flexion exercises are commenced. Over a period of the next few weeks a program of full active, passive, and resisted flexion and extension exercises are gradually introduced.

Other studies have advocated the use of early postoperative active tendon mobilization regimes. These studies have either attempted to increase the strength of tendon repair by altering the suturing technique at the site of laceration (Lahey, 1923; Hernandez et al, 1967; Kessler & Nissam, 1969; Becker et al, 1979), or attempted to avoid tension at the repair site by internally splinting the repaired tendon with a suture that grasps the tendon proximally in the palm, passes through the fibro-osseous tunnel to approximate the lacerated tendon and then passes distally through the distal phalanx (Hester, 1984; MacMillan

et al, 1987). Due to such factors as complicated surgical approaches, high incidence of tendon ruptures and bulky repair sites with these approaches, early postoperative active mobilization regimes have failed to develop wide clinical acceptance.

The concept of early postoperative controlled passive motion following primary flexor tendon repair was first introduced by Young and Harmon in 1960. However, it was not until fifteen years later that this concept was popularized (Kleinert, Kutz & Cohen, 1975). There are two approaches to early controlled passive motion described in the literature.

The first method of early controlled passive motion described, utilizes elastic band traction attached to the finger tip of the repaired digit. This regime is similar to the method described by Young and Harmon (1960) but has been modified by Kleinert et al (1975) and Lister et al (1977). Postoperatively, the hand is immobilized in a dorsal splint that holds the wrists and MCP joints in a flexed position. The splint is fabricated with a dorsal finger portion that allows full IP extension. The rubber band attached to the finger tip is fixated proximally at the wrist and holds the relaxed finger in a flexed position. Commencing on the third to fifth postoperative day, full active finger extension is started and repeated six to eight times, six times daily. The rubber band traction passively flexes the finger following each active finger extension exercise. After three

weeks, the dorsal splint is removed and the rubber band traction remains in place. Active extension exercises are continued, but active flexion is discouraged until six weeks (Cannon & Strickland, 1985).

The second method of early controlled passive motion was first described by Duran and Houser (1975) and later modified by Strickland and Glogovoc (1980). The modification involves a position of rest between exercises of IP joint extension rather than utilizing an elastic band traction that was initially described by Duran and Housar (1975). With this method of early controlled passive motion, the hand is immobilized postoperatively in a dorsal splint that holds the wrist in slight flexion, the MCP joints in 50 degrees of flexion and the IP joints in extension. Commencing on the third to fifth postoperative day, the patient is instructed to twice daily remove the volar finger straps that immobilize the IP's and perform, within the confines of the splint, eight repetitions each of full distal IP, proximal IP and composite (MCP, PIP and DIP) digital passive flexion and extension range of motion exercises. Active flexion and extension exercises are commenced after four and a half weeks, and the splint is discontinued at six weeks (Cannon & Strickland, 1985).

Verification of passive tendon gliding within the fibro-osseous canal when using early controlled passive motion regimes in humans was established clinically by Duran and

Houser in 1975, and later experimentally substantiated and quantified in animals (McGrouther & Ahmed, 1981; Gelbermen, Botte, Spiegelman & Akeson, 1986). Isolated passive joint motion allows independent tendon gliding of the tendon that inserts onto the distal phalanx of the joint being moved. Composite passive digit motion allows maximal gliding of the whole flexor mechanism in relation to the sheath, whereas, combined passive DIP and PIP motion allows maximal differential excursion between the two tendons inserting into the distal two phalanges.

Clinical studies showing improved digital function with early postoperative controlled passive motion (Strickland & Glogovoc, 1980, Kleinert & Verdan, 1983) have helped to establish clinical acceptance of these early postoperative regimes. Experimental studies have also shown some interesting results. In addition to improved tendon excursion (Young et al, 1981), tendons managed in this fashion have also shown evidence of healing through an intrinsic healing process without scar formation (Gelberman et al, 1985; Nelson et al, 1985) and have demonstrated evidence of greater rupture load throughout the third to twelfth (Gelberman et al, 1982) and first to sixth (Hitchcock et al, 1987) postoperative weeks, when compared to tendons managed by early postoperative immobilization.

Early controlled passive motion regimes presently described, delay initiation of active tendon motion longer

than the three weeks that has been traditionally described when early postoperative immobilization is utilized. The reason for this delay in active tendon motion with early controlled passive motion is unclear. Caution, for fear of rupture, has been advocated perhaps on the assumption that tendons managed by controlled passive motion heal more slowly and, therefore, develop tensile strength more slowly than tendons managed by early postoperative immobilization. This has not been adequately explored in the literature. Evidence of greater rupture strength with early controlled passive motion regimes during the late (Gelberman et al, 1982) and early (Hitchcock et al, 1987) postoperative healing periods suggests the opposite may be true.

Tendon healing

Specific mechanisms involved in the process of tendon healing have not been clearly established in the literature. There are two different viewpoints presented. One view expresses the opinion that tendon healing is dependent on an extrinsic healing process of ingrowth of granulation tissue from surrounding tissues. The other viewpoint suggests that tendons have the ability to heal through an intrinsic process by tendon cell proliferation.

The process of extrinsic healing in flexor tendons was clearly described by Potenza in 1962. Potenza (1962) reviewed the histological processes involved during the

initial 128 days of healing in immobilized dog flexor tendons. He described an early process of inflammation followed by a proliferation of granulation tissue from surrounding tissues. He also noted that intrinsic tendon tenocytes remained inactive throughout the total healing period. Mason and Shearon (1932) and Skoog and Persson (1954) had previously described similar processes. However, these studies were not conducted on flexor tendons in a synovial environment.

Lindsay and Thomson (1959), and Lindsay and Birch (1964), described morphological changes of healing flexor tendons during the first four weeks of healing. He outlines a process of epitenon fibroblast proliferation that commences on the second day, followed by migration, proliferation and maturation of these epitenon cells into the central portion of the tendon over the next two weeks. Lindsay and Birch (1964), also described a proliferation of intrinsic endotenon fibroblasts, however, he described their contribution as minimal and not commencing until the third week.

Other authors have since demonstrated experimentally an ability of flexor tendons in a synovial fluid environment to heal intrinsically. This process involves a dominant participation of intrinsic tendon cells in the healing process, and suggests that the ingrowth of the adherent scar from surrounding tissues may not be an essential element of flexor tendon healing.

Lundborg (1976) and Lundborg and Rank (1978), demonstrated intrinsic healing in repaired flexor tendons isolated in the synovial environmental of rabbit knee joints. These same authors, in a later study (Lundborg et al, 1985) were again able to demonstrate this intrinsic healing potential in flexor tendons, without possible contamination of the synovial environment by extrinsic fibroblasts, by sealing repaired flexor tendons in synovial chambers and placing them in the subcutaneous tissues of rabbits' back. Many other authors have also demonstrated experimentally similar intrinsic healing potentials of repaired flexor tendons (Matthews & Richards, 1976; McDowell & Snyder, 1977; Matthews, 1979; Becker, Graham, Cohen & Diegelman, 1981; Graham, Becker, Cohen, Merritt & Diegelman, 1984; Manske & Lesker, 1984; Gelberman, Manske & Vandeberg, 1984).

Ketchum (1977) and Lundborg and Rank (1987), reviewed the processes of flexor tendon healing and concluded that primary tendon healing can occur through either intrinsic or extrinsic healing processes. They state that a key factor in determining the primary direction of the flexor tendon healing towards regeneration (or extrinsic healing) or towards the more primitive reparative process of healing by scar tissue (extrinsic healing), is the nutritional supply of the repaired tendon during the early postoperative healing period.

Nutrition of the flexor tendon in the fibro-osseous tunnel

of the human finger has been demonstrated through both vascular perfusion and synovial fluid diffusion pathways. The extent to which each source supplies the nutritional needs of flexor tendons in the fibro-osseous tunnel of the finger has received considerable study. Manske and Lesker (1985) and Hunter (1987), both provide excellent reviews on work conducted on the vascular anatomy of the flexor tendons in the finger. In summary, the Flexor Digitorum Profundus (FDP) tendon can be described as having two separate intrinsic systems of vascular supply. The proximal vascular supply of longitudinally oriented vessels is derived from paratendinous vessels and the distal vascular supply of complex, segmentally arranged, dorsal vascular loops, derived from specialized mesotenon vascular channels called vincula. The two intrinsic FDP vascular systems become demarcated at the base of the proximal phalanx. The distal intrinsic vascular arcades are found only in the dorsal portion of the tendon and do not pass through the fascicular bundles. As well, the distal intrinsic vascular arcade supply is segmental and highly variable, both between people and within the same person as he or she ages. After the third decade, areas of intrinsic avascularity in the FDP tendon increase.

Uniqueness of the intrinsic vascular arrangement and areas of avascularity within the distal portion of the FDP tendon, have suggested to many investigators that flexor tendons within this region may be more dependent on synovial fluid

diffusion than vascular perfusion.

Manske and associates (1978 & 1983), investigated the uptake of titrated proline through vascular perfusion and synovial diffusion pathways in various experimental animal models (dog, monkey, rabbit and chicken), and demonstrated a predominate role of synovial diffusion in flexor tendon nutrition. A system of longitudinally arranged channels running within the intrafascicular bundles of the tendon described by Edwards (1946) and Brokis (1953), led McDowell and Snyder (1977) to present a synovial fluid diffusion theory for flexor tendon nutrition. These authors noted that the vascular arterial loops were similar to the vascular loops found in synovial membranes of joints and suggested that these vessels produced synovial fluid that could be diffused within the tendon substance. McDowell and Snyder (1977) equated this diffusion process to the dynamic synovial diffusion within the cartilage of joints. They postulated that dynamic diffusion of synovial fluid within the tendon was dependent upon a pumping action generated by the repetitive loading and unloading of the tendon with distraction and relaxation with tensile loading, and a milking action produced when the tendon glides over hard surfaces such as the tendon sheath pulley system.

Weber (1979 & 1987), has since demonstrated experimentally, that tendon motion enhanced the diffusion of fluorescein marked synovial fluid from the avascular dorsal

region of the tendon, into the central region of the flexor tendon. Like McDowell & Snyder (1977), Weber (1987), also postulated a dynamic pumping action for diffusion of synovial fluid within the tendon.

It appears from the literature that both vascular perfusion and synovial diffusion play a role in flexor tendon nutrition in the fibro-osseous tunnel of the finger. The more recent literature suggests, however, that dynamic synovial fluid diffusion plays the predominant role. The concept of motion being an integral component for synovial fluid diffusion is also important when considering the increased nutritional demands of a healing tendon.

Injury and surgical repair can affect the healing potential of the flexor tendon in the fibro-osseous tunnel by interfering with the delicate vascular anatomy in this region (Matsui et al, 1987). Postoperative immobilization of a repaired flexor tendon can further compromise the tendons nutritional supply by diminishing the effectiveness of the dynamic synovial fluid diffusion pump.

One possible mechanism for the reported beneficial clinical and experimental effects of early controlled passive motion on healing flexor tendons, is improved synovial fluid nutrition during the early healing period. Improved nutrition during the early healing period with controlled passive motion, could conceivably influence the early healing process towards a more efficient and/or intrinsic healing

process. With this improved nutrition, examination of specific early tensile properties of healing flexor tendons managed by controlled passive motion should reflect an improved healing potential over immobilized tendons.

Tensile properties of normal tendon

Tensile properties of tendon are measured by a uniaxial tensile test in which the tendon specimen is elongated at a constant rate with changes in length plotted against loading force. The tensile properties exhibited by tendon are related to the structural design of the tissue matrix.

Besides water, which accounts for 2/3 of tendon's overall mass, the primary structural component of tendon is collagen. Collagen constitutes approximately 3/4 of the tendons dry mass. The remaining portion of the tendon tissue matrix is composed of elastin fibers, glycosaminoglycans and other substances such as enzymes, glycoproteins and lipoproteins (Frank, Amiel, Woo & Akeson, 1984).

The tendon's primary function can be defined as a transmitter of tensile forces generated by the muscle to the mobile bony elements. A structural design that facilitates this force transmission is important and the parallel arrangement of densely accumulated collagen fibers within the tendon fulfills this functional need.

Nimni (1983), describes the primary role of collagen as a resister or transmitter of tensile forces and describes the

complex arrangement of organized tropocollagen protein macromolecules in collagen fibers. Each tropocollagen molecule is formed by a triple helical arrangement of complex amino acid chains. The hierarchical arrangement of these tropocollagen molecules first into microfibrils, by an end-to-end and lateral 1/4 staggered aggregation, followed by further parallel aggregation of these microfibrils into collagen fibrils constitutes the primary structural arrangement of collagen. Further aggregation of collagen fibrils into fibril bundles forms the collagen fiber within a connective tissue matrix (Figure 2.2). This hierarchical, parallel arrangement of tropocollagen molecules within the collagen fiber allows for development of an organized arrangement of covalent bonds within and between the tropocollagen molecules (Figure 2.3). Each one of these covalent bonds is extremely strong and provides a significant resistance of the collagen fiber to tensile loads and chemical denaturation (Viidik, 1973).

The further organization of collagen fibers into a densely organized, parallel, hierarchical arrangement within the connective tissue matrix is what distinguishes tendons and ligaments from other connective tissues, and provides them with a structure suited to the transmission and resistance of large tensile forces. Butler, Grood and Noyes (1978), describe the ultrastructure of a tendon. Collagen fibers and a few supporting tenocytes are formed into primary

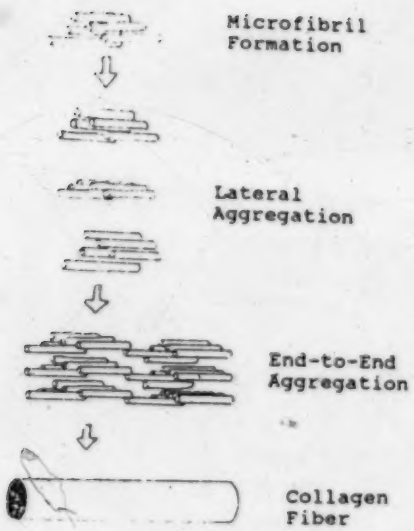

Figure 2.2: Collagen Fiber Microfibril End-to-End and Lateral 1/4 Staggered Aggregation. (Adaped from: Nimni, 1983).

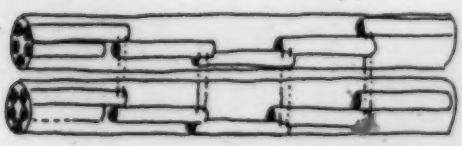

Figure 2.3: Collagen Fiber Intra- and Inter-Fibril Covalent Crosslinks. (Adaped from: Nimni, 1983).

tendon bundles called fascicles and are surrounded a loosely organized connective tissue sheath called the endotenon. The fascicles are then grouped and held together within another loosely organized sheath of connective tissue called the epitenon, which condenses peripherally to form the external surface of the tendon (Figure 2.4). Butler et al (1978), also describe a typical crimped or waveform pattern of the primary fiber bundles found within the tendon. Viidik (1973), states this crimp waveform is caused by the collagen fibers bonding with other tendon tissue matrix macromolecules such as elastin.

A typical load / elongation tensile testing curve for tendon has been described by Butler et al (1978) (Figure 2.5). The uniaxial tensile test curve for tendon has three regions. The initial region has been referred to as the 'toe-region' and is characterized by a curvi-linear (convex towards the x-axis) minimal increase in load as the tendon specimen is elongated to approximately 3%. During this initial region of tendon elongation the tendon collagen fibers are stretched from a resting crimped pattern. If the load is released during this initial elongation region the tendon will behave in an elastic fashion and return immediately to its original resting length.

The second region of the curve described by Butler et al (1978), is represented by a rapid linear increase in load. During this linear region of tendon elongation, the parallel

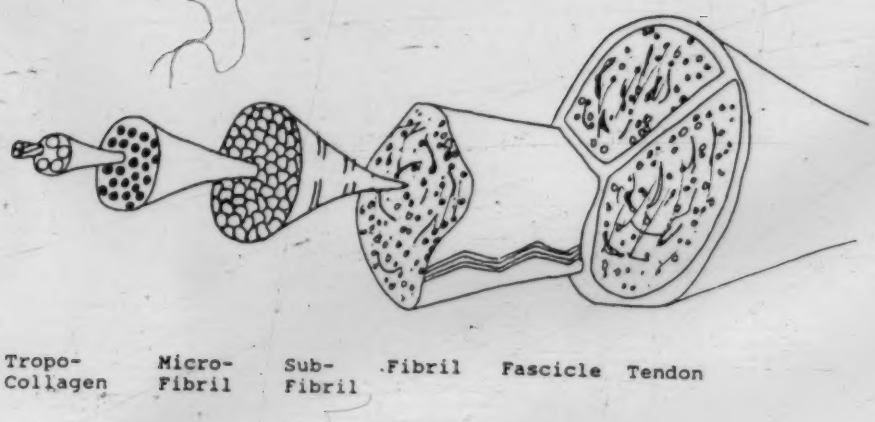

Figure 2.4: Tendon Ultrastructural Arrangement. (Adapted from: Butler, Grood and Noyes, 1978).

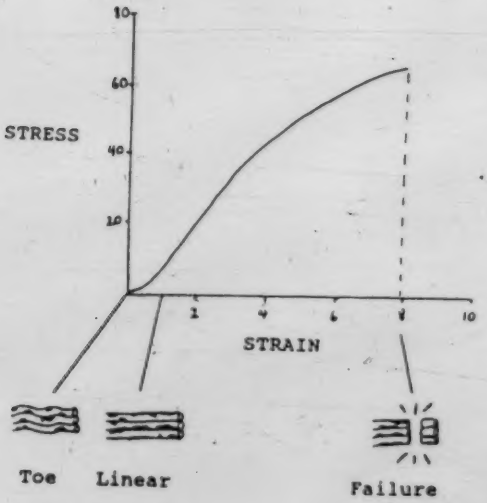

Figure 2.5: Typical Load / Elongation Tensile Curve for Tendon. (Adaped from: Butler, Grood and Noyes, 1978).

oriented collagen fibers are undergoing a physical elongation. Nimni (1983), also describes an actual microfailure of some of the shorter collagen fibers loaded during this region of tendon elongation. If the load is released during this region of elongation, the tendon will behave in a visco-elastic manner and take time to return to its original resting length.

The third region of the curve described by Butler et al (1978), is the failure region. This failure region is characterized by another curvi-linear (convex away from the x-axis) increase in load. Gradually the load bearing capability of the tendon begins to plateau, or decrease slightly, before the tendon specimen fractures. Specific failure and fracture characteristics of tendon have not been described. However, tensile material properties during the initial curvi-linear portion of this failure region have been summarized by Butler et al (1978). The point of change from the second linear region of the curve to the third curvi-linear region is referred to as the yield point. Permanent, or plastic structural changes occur in the substance of the tendon, if the tendon is strained beyond this yield point. If the load is released during this initial failure region, the tendon specimen never returns completely back to its original resting length.

Butler et al (1978), also described the need to standardize the load / elongation curve by dividing the load

by the cross section area of the tendon and plotting this curve against the percentage of elongation of the tendon specimen. This standardization procedure is necessary in order to compare tensile properties between tendons, as the overall structural tensile characteristics of the tendon specimen will change with differences in width and length of tendon specimen tested. This standardized tensile curve is called a stress-strain curve.

Various material tensile properties of the tendon can be determined from the stress-strain curve. Maximum stress and strain to failure, material stiffness and energy absorbed to failure are some of the more common material properties determined from the stress-strain curve. Stress and strain values are determined by extrapolating x and y co-ordinates at the point of failure. Energy absorbed to failure is determined by calculating the area under the curve and a value for material stiffness can be determined by calculating Young's modulus of elasticity (stress/strain) (Butler et al, 1978).

Reported values for normal tendon material properties vary somewhat in the literature. Reported maximum strain values for human tendon range between 10 and 18% (Elliott, 1965 & 1967; Harkness, 1968). Elliott (1967), states that tendon requires a stress of 10kg/mm^2 to acheive a strain of 5%. Harkness (1968), describes a stress value ranging of 5 - 10kg/mm^2 to acheive a strain of 6%. Yamada (1970), describes

a maximum stress value of 50 - 100N/mm^2 (5.1 - 10.2 kg/mm^2). Gordon (1984), describes a maximum stress of 82N/mm^2 (8.4kg/mm^2).

Reported values for specific material properties of tendon are somewhat misleading, as these values may vary depending on such things as tensile testing strain rate, environmental conditions, technical problems with gripping, cross sectional area and strain measurement procedures and differences in the age, sex and activity level of the donor specimen.

Tensile material properties for tendons vary due to differences in strain rates, as tendons are a composite material that is primarily visco-elastic in nature. Visco-elastic materials demonstrate time-dependent material properties. Some of these time-dependent characteristics (Viidik, 1973; Butler et al, 1978) are:

1. Force relaxation, in which a constant elongation maintained over a period of time causes the tendon to demonstrate a progressive decrease in the load required to maintain the elongation.
2. Creep, in which a constant load is applied over time and the tendon demonstrates a progressive increase in elongation.
3. Strain rate sensitivity, in which maximum stress, energy absorbed to failure, and material stiffness increase as the rate of tendon elongation increases, whereas, maximum strain decreases. This functional

characteristic of strain rate sensitivity in tendon, means that tendon becomes more stiff and behaves in a more elastic manner when it is strained at a fast rate. At fast strain rates tendons develop greater strength capabilities but lose their capacity to withstand significant deformation.

Variations in environmental testing conditions will also affect the material properties of tendon. Changes in the environmental temperature and relative humidity have been shown to alter the material properties of the tendon being tested (Galante, 1967; Warren, Lehmann & Koblanski, 1971; Viidik, 1973). Methods of preservation of tendon have also been shown to alter the tensile properties of tendon. Embalmed tendons show a significant increase in tensile strength (Blanton & Biggs, 1970), whereas, tendons that have been frozen have shown no significant change in their material properties (Ridge & Wright, 1965; Galante, 1967).

Viidik (1973), reviews some of the technical difficulties encountered when trying to test a slippery, flexible, compressible material such as tendon. The major hurdle to overcome in testing a fresh tendon specimen is designing a clamp that holds the specimen firmly enough to avoid slippage, but not so firm as to cause specimen failure in the clamp. Some modifications to clamps described by Viidik (1973), have been the use of a wedged clamp that graduates

the pressure within the grips, wrapping the tendon with wire or surfacing the clamp jaws with a rough material to increase the coefficient of friction between the tendon and the clamp, and rounding of the clamp jaw edges to avoid a stress concentration at the edge of the clamp jaw.

Other technical difficulties are encountered when trying to measure the cross-sectional area and strain of the tendon specimen. Ellis (1968), discusses the reliability and validity of different cross sectional area measurement techniques (caliper measures, histological section, dry specimen weight/unit length and shadow contour reconstruction). Ellis (1968), concluded that a shadow contour reconstruction method was the most valid approach for direct measurement of a fresh, moist, compressible specimen. However, the reliability of the shadow contour method in his investigation was less than was a measurement of dry specimen weight/unit length. A problem discussed by Viidik (1973), with the validity of dry specimen weight as it correlates to cross-sectional area is that it is only appropriate when the specific gravity of the total specimen is constant.

Viidik (1973), also discusses the difficulty with determining an initial strain value for a tendon when the strain of the specimen is indirectly determined by measuring the distance between the two clamps. This approach is only valid if there is no slippage of the tendon specimen in the grips and the specimen is preloaded to a standardized value

prior to the commencement of the test. As well, with the commencement of the test, there is a time delay during which the compliance in the testing apparatus is absorbed prior to the test specimen being loaded at a constant strain rate (Vrijhoef & Driessens, 1971). Therefore, the initial portion of the load / elongation curve is representative of this machine / specimen interaction and is not truly representative of the material properties of the tendon being tested.

Other authors have demonstrated that the tensile properties of tendon vary with the age, sex and activity level of the specimen donor. Noyes and Grood (1976), demonstrated that older ligament specimens show a lower stiffness and maximum stress and strain than do younger specimens. Tipton, Matthes, Maynard and Carey (1975), demonstrated that tendons and ligaments were stronger and larger following long term physical activity. As well, hormonal changes with aging and sex differences have also been shown to affect the material properties of normal tendon (Booth & Tipton, 1970).

From reviewing the literature in the area of normal tensile properties of tendon, it can be seen that there is great variability in the reported tendon tensile material properties reported in the literature. These reported differences may be due to such factors as different strain rates, environmental testing conditions, testing procedures

and differences in the age, sex and activity level of the donor specimen in the various studies examined. Attempts to control factors that may affect tensile properties of tendon should be included and discussed in any study examining tensile properties of tendon.

Tensile properties of healing tendon

Tensile properties of healing tendon have received limited experimental investigation. Mason and Allen (1941), in their classic study on dog extensor carpi radialis tendons, were the first to investigate tensile strength gains of healing tendons. They defined three phases in tendon healing as determined by tensile strength changes. The first phase lasted five days and was characterized by a rapid diminution in strength. The second phase demonstrated a progressive increase in tensile strength which reached a plateau at about sixteen days. The third phase showed a secondary rise in strength commencing on the 19th to 21st day. These same authors also note that three phases of tensile strength gains correlated with the three phases of connective tissue healing (inflammation, fibroplasia, maturation). As well, Mason and Allen (1941), describe an increase in scarring and adhesions in healing tendons that commence active tendon mobilization prior to three weeks, but a significant improvement in rupture strength gains in tendons that commence active tendon motion after three weeks of immobilization.

Madden and Peacock (1971), examined healing dermal connective tissue wounds and demonstrated a correlation during the initial three weeks of healing between the accumulation of collagen in the wound and its breaking strength. However, after three weeks the collagen content stabilized and the breaking strength of the wound continued to rise. These authors attributed this secondary rise in strength to a secondary remodelling and maturation of the collagen matrix in the scar.

Hirsh (1975), examined tensile strength gains in healing rabbit achilles tendon and demonstrated that the breaking strength of immobilized tendons did not exceed maximal muscle power until 8 weeks postoperatively and reached only 50% of normal strength at 24 weeks. Findings from this study suggest that healing immobilized tendons develop strength slowly and support the contension of Peacock et al (1971), that resisted muscle work of the immobilized tendon should be approached with caution.

Goldin (1980), conducted an excellent study on immobilized healing rabbit tendon. Goldin (1980), concurrently measured biomechanical, biochemical and structural changes in healing tendons between the 5th and 10th days of healing. The author described a histological process of extrinsic scar proliferation across the repair gap. As well, he measured isometric thermal shrinkage properties of the healing tendon and assessed hydroxyproline, uronic acid and hexosamine

levels of the healing tendons. Goldin (1980), concluded that the physical and mechanical changes during the 5th to 13th day were found to occur concurrently with the biosynthesis of both collagen and tissue glycosaminoacid (GAG) content of the healing wound. He felt that the restoration of form and function of the healing tendon depended on the total integration of all the structural components of the healing tendon and not just the collagen content of the wound.

Forrest (1983), in his review article on connective tissue healing states that early tensile strength gains of healing scar tissue has little to do with the formation of immature collagen fibers in the wound. Forrest described a network of myofibroblasts, reticulin, and fibronectin in the early healing wound granulation tissue that he felt could be responsible for early tensile strength of healing connective tissue wounds.

It appears from these reported investigations on tensile strength gains in healing tendons, that immobilized tendons demonstrate a slow increases in tensile strength, especially during the initial three postoperative weeks. What has not been clearly described is what factors, or tissue components, contribute to the early tensile strength gains in healing tendons.

Some reports in the literature suggest that the application of limited tensile stresses across a healing connective tissue wound will enhance its tensile strength

gain. Mason and Allen (1941), were the first to note that active tendon motion commenced at three weeks significantly improved the rupture strength in healing tendons. Thorngate and Ferguson (1958), examined the effect of tensile stress on healing aponeurotic wounds in rabbits. These authors noted that tension produced orientation of collagen fibers in the direction of stress in aponeurotic wounds healed under tension between the 7th to 21st days, and that wounds healed under tension had a greater bursting strength than did wounds that did not heal under tension.

Farkas, Herbert and James (1979), investigated the effects of intermittent active tendon mobilization commenced on the 18th day on the healing strength of chicken flexor tendons. Although, these authors found no difference in tensile strength at 35 days between tendons that commenced active motion at 18 days as compared to 25 days, they were able to demonstrate a four times greater tensile strength at 35 days for both active tendon mobilization groups when compared to the continuous immobilization group.

Gelberman et al (1982), compared the maximal tensile load at failure of healing canine flexor tendons, between tendons managed by postoperative immobilization and tendons managed by an early controlled passive motion regime. These authors demonstrated a significant difference between the two management regimes in maximal load at failure during the 3rd to 12th postoperative weeks. Unfortunately, these authors

only examined the overall rupture load of the healing tendon during the later stage of scar maturation. Differences in specific tensile material properties during the earlier fibroplasia phase of tendon healing were not examined.

Becker and Diegelmann (1984), demonstrated enhanced intrinsic tendon fibroplasia and fibroblastic orientation along lines of tension in an in vitro examination of healing flexor tendons during the initial 14 days of healing. Gelberman et al (1985) and Nelson et al (1985), also demonstrated histological evidence of an enhanced intrinsic epitenon fibroblast proliferation and migration in repaired flexor tendons during the initial two postoperative weeks, for healing flexor tendons managed with an immediate postoperative controlled passive motion regime. Findings from these histological studies suggest that early tensile stresses may facilitate the intrinsic healing potential of flexor tendons.

A more recent study by Hitchcock et al (1987), published after the completion of this study, examined the effect of immediate constrained digital active and passive motion on the overall rupture tolerance of healing chicken flexor tendons during the initial five to forty day healing period. These authors demonstrated a significant difference in rupture load throughout this early postoperative healing period, between tendons managed by postoperative immobilization and early postoperative constrained active and

passive motion. Findings from this study provide support for the effectiveness of early controlled stresses across healing flexor tendons in increasing the overall rupture tolerance of the tendon during the early fibroplasia phase of healing. Unfortunately, these authors did not examine the other material properties of the healing tendons during this early postoperative healing period.

Bassett (1971), in an extensive review article on the effects of stress on connective tissue, describes four factors that may be significant when considering the effect of stress on healing flexor tendons. First, he cited his own work (Bassett, 1962), in which he stated that all connective tissues are derived from the same undifferentiated mesenchymal cells and that their pathway of development depends on the microenvironment that the cell is exposed to. In this study he exposed mesenchymal cells to compression or tension forces at high or low oxygen tensions and found that compression and high oxygen produced bone, compression and low oxygen produced cartilage and tension and high oxygen produced fibrous tissue.

A second factor described by Bassett (1971), is the role physical forces play in the orientation of connective tissue macro-molecules. Bassett (1971) noted that Gayda described electric potentials in connective tissues as early as 1912 and postulated that these electric potentials were due to piezoelectricity. Piezoelectricity is produced when some

crystalline-like materials are stressed. Collagen is crystalline in nature and has the potential to produce these electrical potentials when stressed. The electrical potential or charge produced is proportional to the magnitude of deformation of the crystal. Bassett (1971) again cited his own work (Bassett, 1962 & 1964), in which he demonstrated that weak electrical currents passed through connective tissues caused orientation of the collagen fibers in the direction of the force potential.

A third factor described by Bassett (1971), relates to a postulated 'auto-control' mechanism within the connective tissue. Bassett (1971) felt that stress induced electrical charges on the surface of collagen fibers directed the aggregation and eventual maturation of collagen fiber bundles within the connective tissue by selectively repelling or attracting adjacent collagen fibers.

Finally, Bassett (1971) cites the work of Anderson (1968), in which the author noted the presence of motion induced streaming potentials within tendon. Bassett (1917) postulated that these streaming potentials are generated by hydraulically induced charge separations and represent a massaging of body fluid through the extracellular matrix of the hypovascular tendon tissue. Bassett (1971), concluded by stating that all four of these factors described were evidence that both nutritional factors and physical forces determined in part the direction of cell specialization and

behavior within connective tissues.

Findings of the beneficial effects of stress on healing connective tissues, suggest another possible mechanism for the reported beneficial effects of early postoperative controlled passive motion on healing flexor tendons. If early controlled passive motion regimes following primary repair of flexor tendons within the fibro-osseous tunnel produce a tensile force across the healing tendon callus, then this force should:

1. Stimulate the production, orientation and aggregation of collagen fibers within the tendon callus along the lines of stress-induced piezoelectric forces.
2. Enhance the diffusion of synovial fluid with the healing tendon by generating streaming potentials within the tendon.

Both of these factors mentioned above should enhance the early healing potential of a flexor tendon managed by an early postoperative controlled passive motion program and, therefore, should also be reflected in an examination of the specific material tensile properties of the healing tendon.

Chapter III

MATERIALS AND METHODS

Experimental design

A 2 x 5 (treatment condition x healing period) factorial design, with one between subject factor (healing period: 10, 15, 20, 25, and 30 days) and one within subject factor (treatment condition: immobilization vs controlled passive motion) was utilized in this study (Edwards, 1972).

Thirty experimental white Leghorn chickens were initially assigned at random to one of five groups. Designation of treatment foot and immobilization foot was alternated between chickens within these inital five groups. The five initial groups were then randomly assigned to one of five designated healing periods (10, 15, 20, 25 and 30 days) (Figure 3.1). In this experimental design, each chicken acted as its' own matched control for the experimental treatment conditions (immobilization vs controlled passive motion) under study.

Pre-operative management

Chickens were housed and maintained in individual (18" x 24" x 24") cages, one week prior to surgery, in the small animal care facility of the Surgical/Medical Research Institute (SMRI) on the seventh floor of the Dentistry/Pharmacy Building on the University of Alberta campus. This one week acclimatization period allowed the

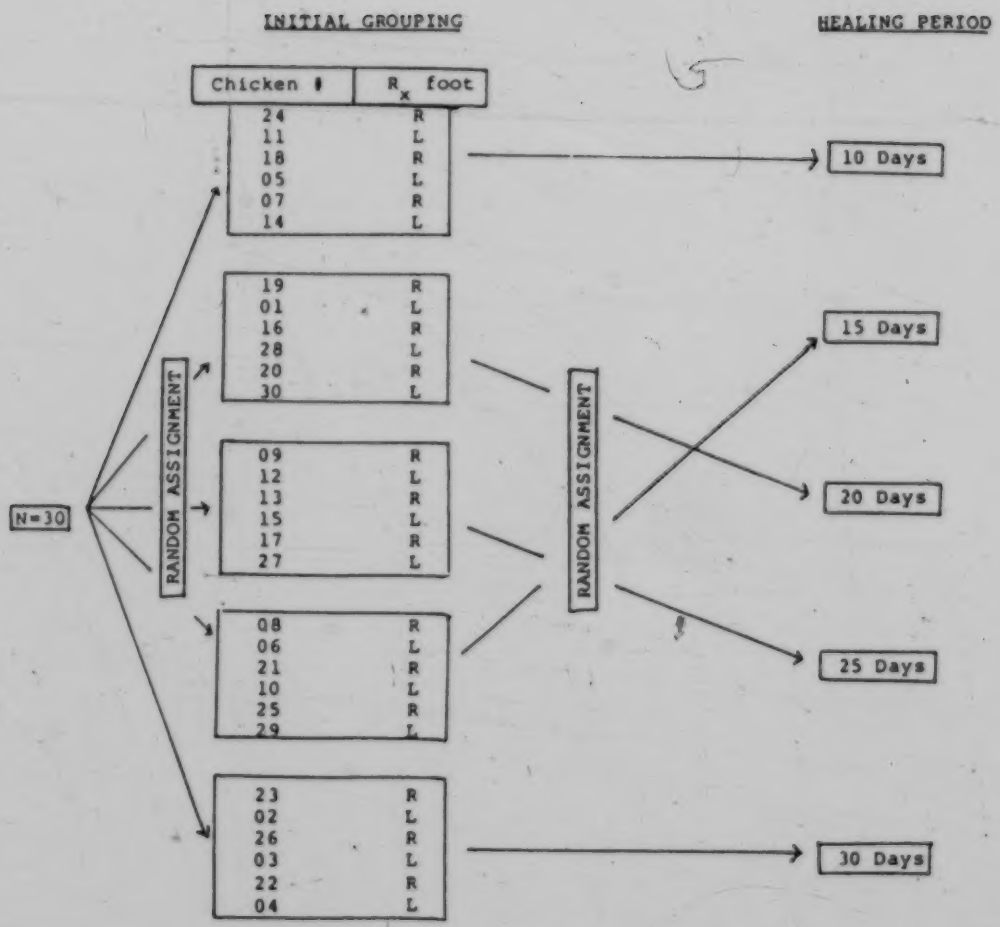

Figure 3.1: Experimental Design Flow Chart

chickens the opportunity to become accustomed to their new environment and also controlled, to a certain extent, the activity level of each chicken prior to surgery.

Chickens previously purchased and awaiting surgery, were housed and maintained as a group at the University of Alberta, Ellerslie farm. SMRI provides seven day per week animal care in compliance with the guidelines of the Canadian Council on Animal Care and the regulations enacted under Section 50 of the Universities Act.

Each individual cage was made of stainless steel and had a metal grid floor. The cages were contained in one room (10' x 12') on three mobile carts that could hold up to six individual cages each.

Each chicken had free access to food (Masterfeed, 16% protein poultry pellets) and water (containing Nutrifurizone Powder, NF). During the one week acclimatization period the chickens were handled twice daily. During the handling the chickens were weighed, given water (approx. 10 cc) and fed water soaked pellet food balls (approx. 50 grams). Supplemental feeding was included to ensure adequate nutrition during the acclimatization and early post-operative periods, as the chickens tended to respond to the change in their environment by becoming lethargic and sleeping most of the day. Plates 3.1 and 3.2 demonstrate the supplemental feeding activities. Chickens were also monitored closely for any signs or symptoms of disease during this one week

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Plate 3.1: Supplemental Feeding

Plate 3.2: Supplimental Water

acclimatization period.

One day prior to surgery, each chicken was fitted with bilateral custom made thermoplastic splints and plaster casts to immobilize the feet and hock joints and then placed into a custom made foam-padded suspension jacket.

A position of postoperative immobilization for the chicken leg was established, that replicated the Duran-Housar / Strickland postoperative immobilization position in humans (wrist flexion, metacarpal phalangeal flexion, and interphalangeal extension). Postoperative immobilization relieves tension across the repair site by bringing the flexor muscle/tendon mass into a shortened position. In order to replicate this shortened and immobilized position in the chicken it was necessary to devise a system that immobilized the hock (ankle) joint in 60° of plantarflexion, the metatarso-phalangeal (MTP) joints in full flexion and the inter-phalangeal (IP) joints in full extension. See Figure 3.2, for a comparison of the positions of postoperative immobilization in a person and in a chicken.

The hock joint was immobilized with four layers of fast drying plaster casting material (Gypsona) wrapped over a double layer of cotton gauze (Tubinet, size 12; Seton Ltd., Oldham, England). The cast extended from approximately the distal 1/4 of the calf to the proximal 1/4 of the metatarsal bone.

The foot was immobilized in full MTP flexion and IP

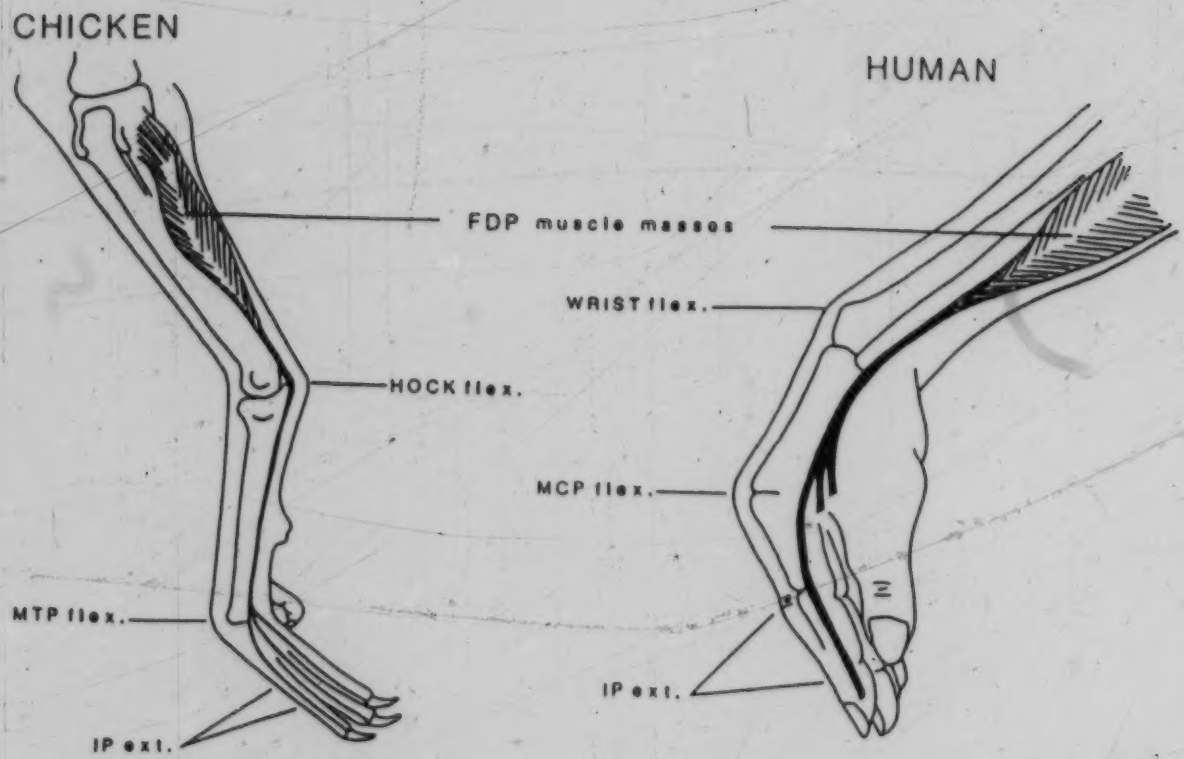

Figure 3.2: Comparison of Postoperative Immobilization Positions Between the Experimental Chicken Animal Model and the Human.

extension with custom fit thermoplastic splints (San-Splint XR; Smith & Nephew Inc., Lachine, Quebec). The splints were lined with .3cm thick adhesive padding (San-liner; Smith & Nephew Inc., Lachine, Quebec). The splint fit around 2/3 of circumference of the distal 1/2 of the metatarsal, and incorporated a hole for the dew claw. This ensured the splint would not rotate out of position. The splint then extended dorsally over the toes and held the toes in MTP flexion and IP flexion. The splint was maintained in position by removable, reuseable, one inch self adherent elastic wrap (Coban self adherent wrap; 3M, Surg/Med Division, St. Paul, MN). Plate 3.3 shows the cast and splint immobilization of the hock and foot.

With this position of immobilization for the hock joint and foot, an orthotic system was unable to be established that both allowed the chicken the ability to safely ambulate and sleep. It was found that as long as the hock joint was immobilized in plantarflexion, the chickens were unable to safely maintain their balance while weight bearing through the lower extremities. Therefore, a system of 4-point suspension that prevented weight bearing through the lower extremities was devised (Plate 3.4). This 4-point suspension system provided for a safe and stable means of lower extremity postoperative immobilization, while at the same time allowing the chicken free mobility of its head, neck, wings and legs.

Plate 3.3: Cast and Splint Immobilization

Plate 3.4: Chicken in 4-point Suspension

Suspension jackets were fabricated out of a double layer of densely woven linen/cotton material. The jacket was patterned with holes for the legs and wings, and was designed so that the portion which fit around the abdomen and pelvis of the chicken was padded with a layer of one-inch dense foam, quilted between the two layers of cloth. This same portion of the jacket had holes along the dorsal edges so that a shoelace could be laced through to allow for a custom fit around each chicken. An additional pad of two-inch low density foam padding was placed along the inner central portion of the jacket to provide extra padding for the breast and pelvic bones (Figure 3.3). The portion of the jacket around the thorax was tacked together loosely above the wings and then continued as two dorsal flaps. On the four corners of these flaps, strings with metal clips were attached as a means of providing a 4-point suspension system.

Once fitted with the casts, splints and suspension jacket, the jackets containing the chickens were suspended from the top grid of the cage. Food and water containers were placed within easy access. During this 24 hour pre-operative period, chickens were monitored closely for any signs of distress or discomfort and all appropriate adjustments to the suspension system were made.

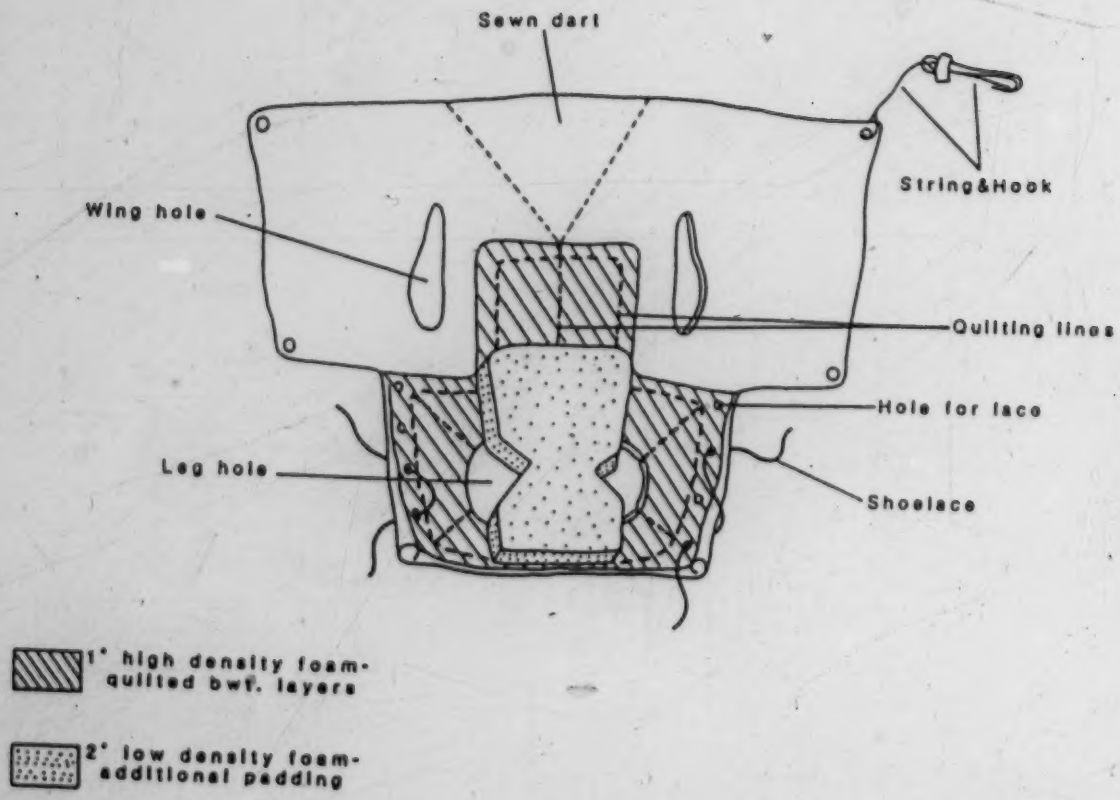

Figure 3.3: Chicken Suspension Jacket Design

Surgical procedure

Immediately prior to surgery each chicken was weighed and gently wrapped with a towel and tensor bandage to immobilize the wings. The feet were then scrubbed with surgical soap, rinsed, and then re-scrubbed with a betadine wash. The two lateral toes were flexed and held in position by a Coban wrap to keep them free of the surgical field. The chicken was then placed on the surgical table. The table was partially covered with an additional towel and surgical drape to provide padding for the chicken. The chickens head was covered by two layers of sterile surgical draping to induce roosting. The drape was loosely fitted around the head to provide adequate air for respiration. This procedure induced roosting immediately, and the chickens slept through the rest of the surgical procedure without the need for sedation or general anesthetic.

Once the rooster was sleeping, the middle toe of each foot was thoroughly prepped with a betadine scrub. A penrose drain tourniquet was clamped on the distal 1/4 of the metatarsal. The middle toe was injected at the level of the metatarsal phalangeal joint along the medial neurovascular bundle with 1% lidocaine with adrenalin (1/100,000) local anesthetic. The foot was then draped so that the middle toe was the only exposed part within the surgical field.

With the aid of 4.5x loupe magnification, a 15-blade scalpel was used for a medial midlateral incision that

extended proximally from just distal to the middle interphalangeal joint to the base of the second phalanx. The neurovascular bundle was identified and retracted volarly and the sheath was opened with a longitudinal incision just volar to the second phalanx and middle interphalangeal joint. Cautery was used up to this point to ensure a bloodless field.

At the level just proximal to the middle interphalangeal joint the volar Flexor Digitorum Sublimus (FDS) tendon, tendon sheath and overlying skin were retracted to ensure adequate surgical exposure. The Flexor Digitorum Profundus (FDP) tendon was then surgically divided with a new 15-blade. The divided tendon was then repaired with a 5-0 Surgilene suture with a sharp (SBE-2) cutting needle. A modified Kessler tendon suture was used for the repair (Strickland, 1985). Each tendon was consistently repaired with the suture first passing through the distal stump and both segments of the repair in the two tendon ends incorporated approximately 1 centimeter of tendon. The repair was finished by firmly approximating the tendon ends and tying the repair with six knots. Following repair of the tendon, the skin was closed with a 4-0 Surgilene suture.

Following skin closure, the middle toe nail was cut just distal to the toe pad and dressed with a small piece of Gelfoam (Upjohn Co., Don Mills, Ontario) and adhesive tape. It was necessary to trim the nail to ensure full passive

digital flexion. The toe had to be trimmed under tourniquet due to small vessels found in the proximal nail. Once the nail was trimmed and dressed, the tourniquet was released and the incision was dressed with a light compressive dressing that consisted of a strip of Sofra-tulle (Framycetin Sulphate BP 1%, Roussel Canada Inc.; Montreal, Quebec), two layers of cotton gauze and Coban. The foot was then placed into the previously fabricated thermoplastic splint and wrapped again in Coban keep the splint in place.

The surgical procedure was then repeated on the contra-lateral foot. For each chicken, both surgical procedures took a combined time of approximately 45 minutes. Following the second surgical repair the drapes were removed from the chickens head. It took approximately 30 seconds for the chicken to fully wake up from the 45 minute roosting state. The chickens were then immediately given approximately 5cc of water and placed on a padded table and monitored closely for approximately 15 minutes to ensure there were no adverse side effects to the surgical procedure.

In this study attempts were made to control for age, site and type of injury, extent of damage to the vincula and sheath, and the skill and surgical technique of the hand surgeon. As well, attempts were made to replicate the ideal surgical repair of a clean Flexor Digitorum Profundus laceration in zone II of the finger.

The controlled surgical factors were:

1. All chickens were of similar age and skeletal maturity.
2. The level of tendon division was approximately 1 centimeter proximal to the volar plate of the middle interphalangeal joint, immediately proximal to the vincula to the profundus tendon at this level (Figures 1.2 and 1.3). This surgical division replicated a clean flexor digitorum profundus tendon laceration at the distal 1/4 of the proximal phalanx, just proximal to the proximal interphalangeal joint and distal to the bifurcation of the flexor digitorum sublimus tendon.
3. All tendons were divided and repaired by the same qualified hand surgeon [J. G. Beauchene, MD, FRCS(C)].
4. All tendons were approached utilizing the same midlateral incision, and repaired with a modified Kessler suture (Strickland, 1985). The initial pilot study surgical trials, showed that a tidy smooth tendon repair could be achieved with just a modified Kessler suture in the small chicken tendon. Therefore, a running epitendinous suture was not done, as suggested by Strickland (1985), as this was felt to introduce unnecessary additional surgical trauma to the tendon.

5. A separate surgical closure of the tendon sheath, as described by Lister (1985), was not utilized in this study. It was again felt, as a result of the pilot study surgical trials, that there was sufficient adherence between the volar skin and tendon sheath by thick fibrous connective tissue bands, that with appropriate skin approximation there would be adequate approximation of the incised flexor sheath edges to allow healing.

Postoperative management

All chickens were placed back in suspension in their individual cages within 1/2 to 1 hour following surgery. During the first 24 hours the chickens were monitored closely and fed and watered at least twice more during the same day.

During the second and third postoperative days the chickens were handled twice daily. The chickens were taken down from suspension for at least 15 minutes, weighed, fed and given water. As well, on the third postoperative day the original postoperative dressings were removed and the surgical incisions examined. The original postoperative dressings were then replaced by a lighter compressive dressing that consisted of a single strip of Sofra-tulle held in place by Coban. This lighter elastic dressing allowed for full passive digital range of motion.

Commencing on the fourth postoperative day a twice daily

five minute session of controlled passive motion was commenced. The controlled passive motion sessions were separated by at least eight hours, and were completed before nine o'clock in the morning and not commenced again until after 5 o'clock in the evening.

A modification of the Duran/Housar-Strickland technique of controlled passive motion was utilized in this study. Validity of a Duran/Housar-Strickland technique of early controlled passive motion in the chicken was verified for the purposes of this investigation, in a pilot study of x-ray excursion of imbedded metal wires in the Flexor Digitorum Profundus and Sublimus tendons at the level of surgical repair. Lateral views of different treatment manipulations in the chickens' foot were taken for three different non-experimental chickens. Two of the chickens' feet were studied 24 hour after surgical imbedding of wires and one was reviewed immediately after surgery.

A measure of the distance of the imbedded wires from a line drawn perpendicular to the axis of the head of the second phalanx with the foot in the position of immobilization, relative to the wires position from this same point of reference with each of the passive joint manipulations was determined (Figure 3.4). This measure of wire excursion at the level of repair gave an indication of the relative motion of the flexor tendons, in relation to the head of the second phalanx, with passive joint manipulations.

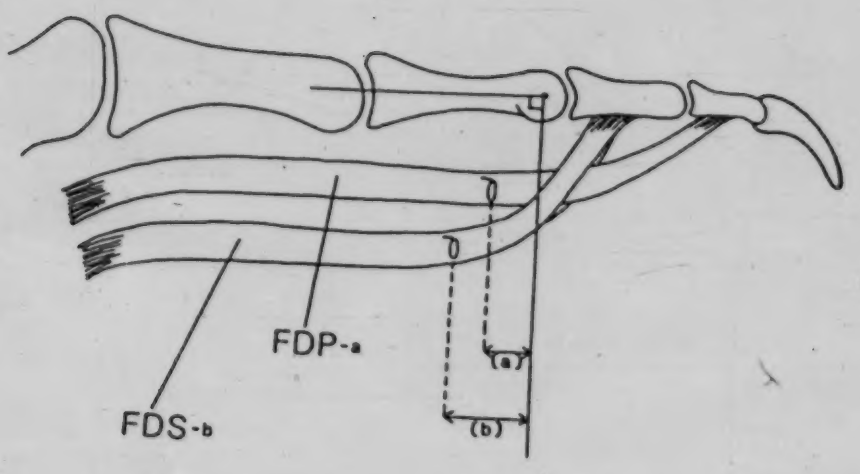

Figure 3.4: X-ray Excursion Study - Method of Measurement of Imbedded Wires.

During each controlled passive motion treatment sessions the chicken was placed on the weight scale and the Coban dressing around the toe portion of the splint was removed to allow access to the middle toe. A metronome was used to establish a rate of one stroke per second. This standardized rate of manipulation was used to ensure a standardized treatment regime between treatment sessions and between chickens. For each one second passive joint manipulation, one complete excursion through flexion and extension was done. Within the five minute treatment session, a total of 300 passive joint manipulations were completed.

The five minute treatment session was divided into six different passive range of motion manipulations. The six controlled passive motion manipulations utilized in this study were: (Figure 3.5)

1. Thirty seconds of isolated distal interphalangeal (DIP) joint flexion and extension.
2. Thirty seconds of isolated middle interphalangeal (MIP) joint flexion and extension.
3. Thirty seconds of isolated proximal interphalangeal (PIP) joint flexion and extension.
4. One minute of combined DIP and MIP joint flexion and extension.
5. Thirty seconds of combined DIP and MIP joint flexion and extension with the PIP joint blocked in flexion.

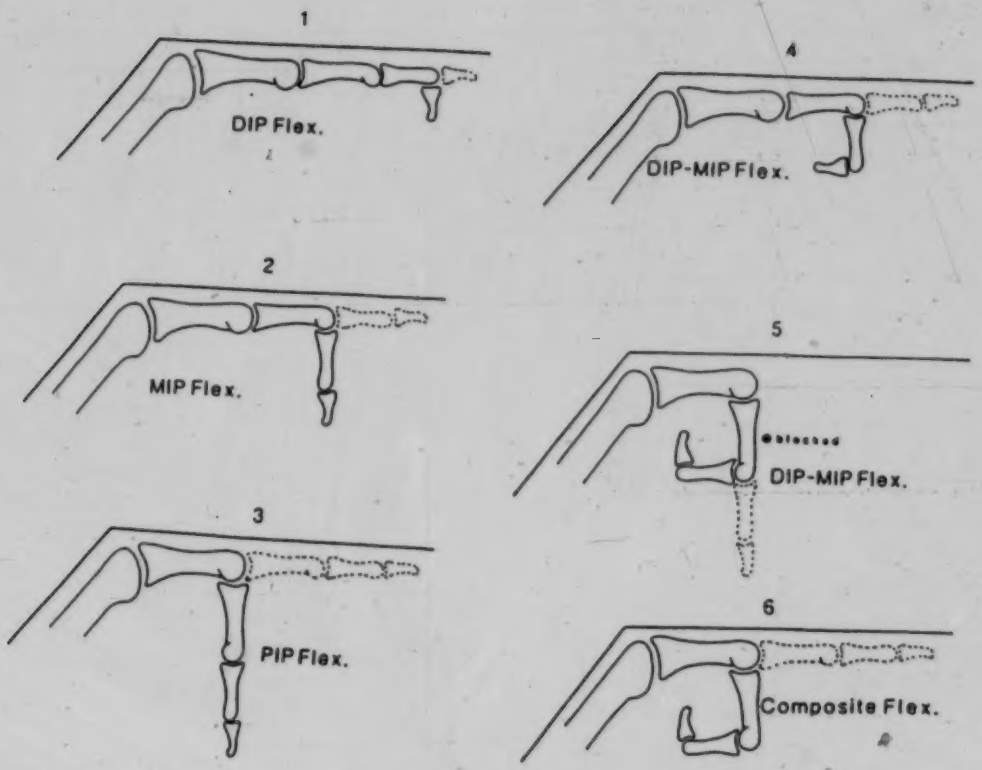

Figure 3.5: Passive Joint Range of Motion Manipulations.

6. Two minutes of composite DIP, MIP and PIP flexion and extension.

Following treatment, chickens were weighed, fed, given water and then placed on a padded towel on the floor for an additional 10 minutes. The additional 10 minutes gave the chickens at least a 15 minute rest from the suspension system. Chickens continued with this twice daily handling and treatment session until sacrifice.

Sacrifice and dissection procedures

On the specified day of sacrifice each chicken underwent the morning handling and treatment session as usual. At the time of sacrifice each chicken was first weighed and then snugly wrapped with a tensor bandage around its wings. The chickens were sacrificed by a quick snap and wringing of the neck.

The suspension jacket was then removed and the chickens were examined for any pressure areas. The order of foot dissection was consistent between chickens, with the right foot always dissected first. The splint and compressive dressings were removed carefully. The cast along the metatarsal was split laterally to allow access to the whole metatarsal. A longitudinal incision along the lateral aspect of the tarso-metatarus was made to expose the underlying flexor tendons. The skin and thick common flexor sheath were

retracted and the common FDP tendon was identified. The FDP tendon was found adjacent to the bone and was easily identified as it was always joined to the tendon of the Flexor Hallicus Longus by thick inter-tendinous bands. These inter-tendinous bands were severed and the FDP tendon was followed first to the hock joint and severed and then followed distally to just proximal to the MTP joints. At the level just proximal to the MTP joints the FDP tendon divides into three separate tendons that continue distally into each of the lateral three digits. The tendon passing to the middle toe was identified and the remaining two FDP tendons were severed at the point of division from the proximal common FDP tendon. The lateral metatarsal incision was then irrigated with saline and left.

Dissection at the level of the toe was then carried out. The sutures were removed and the original surgical incision was re-opened and extended proximally to the MTP joint and distally to the lateral nail bed. The volar skin along the whole length of the toe was then carefully separated from the underlying flexor sheath and removed. The sheath was opened by cutting along the whole length of the volar surface of the tendon sheath with sharp curved scissors. The sheath was reflected and the two superficial flexor tendons [Flexor Digitorum Perforatus (FDPe) and Flexor Digitorum Sublimus (FDS)] were also cut longitudinally commencing at the bifurcation of the FDS tendon and progressing proximally

through both tendons to the level of the planter fat pad. The two superficial tendons were then completely severed from their more proximal portion and carefully reflected from the under lying FDP tendon in the toe.

The distal insertion of the FDP tendon was then severed and dissection of the FDP tendon progressed proximally. The underlying vincula at the level of the DIP joint were cut and the distal end of the FDP tendon was carefully reflected proximally. This procedure exposed the dorsal surface of the tendon to the point of surgical repair. The vinculum to the FDP tendon just distal to the repair site was identified and cut. This procedure freed the FDP tendon except for any adhesions at the repair site. Any adhesions of the tendon callus to the underlying bone or lateral slips of the FDS tendon were carefully removed with blunt dissection, ensuring that the healing tendon callus was not disrupted.

The freely dissected FDP tendon was then lifted from the chickens' foot, placed on a piece of 1cm thick, dense cork sheeting and irrigated with saline. The tendon was held in place on the cork by two bent pins at either end of the tendon. An identifying label was also attached to the cork. The cork, label and dissected tendon were then immersed in liquid nitrogen.

Following dissection and immediate freezing, the tendons were stored at -80^{o}C in a Revco Ultra-Low freezer (Rheem Manufacturing Co., Scientific Products Division; Ashville,

NC). However, prior to storage, the tendons were removed from the liquid nitrogen and the identifying label was removed. The cork and tendon were then wrapped tightly in a double layer of Saranwrap. A further layer of double thickness 4x8 cotton gauze was wrapped around the Saranwrap and then snugly taped with 3 inch adhesive tape. The identifying label was then re-attached to the outside of the tendon bundle and the bundle was stored in an air tight styrofoam container in the Ultra-Low freezer.

Following the completion of dissection and storage of all tendons, the identifying labels on the tendon bundles were removed by an independent assistant and replaced with a number written on the outside of the tendon bundle with felt pen. This procedure ensured that tensile testing and initial data analysis would be carried out under a blinded condition. The identifying labels and corresponding number records were kept in a sealed envelope and were not made available to the author until the analysis of the tensile testing pen plot load/elongation curve for all tendons was complete.

Tensile testing procedure

Frozen tendon bundles were transported for tensile testing in liquid nitrogen to the materials testing laboratory in the Chemical/Mineral Engineering Building. Tensile testing of all tendons was completed in two consecutive days.

Tendons were removed from the liquid nitrogen in groups of

six. Tape/gauze/saran coverings were removed and the cork and tendon were placed in room temperature saline to thaw for approximately one minute. The tendons were removed from the cork and placed on a saline soaked paper towel beside the appropriate identifying number. All six tendons were then covered with another layer of saline soaked towel and taken for cross-sectional area measurements.

Cross sectional area measurements were taken using an optical comparitor (Scherr Tunico, Model 22-1500) (Plate 3.5). The optical comparitor reflected a magnified (10x) image of the tendon specimen onto a translucent glass screen (Plate 3.6). The screen had vertical and horizontal lines to allow for orientation of the reflected tendon specimen. Tendon specimens were placed in a saline filled petri dish on the optical comparitor platform and held taut for measurement of the width of the tendon callus. A width measurement of the tendon callus at the level of repair was made with digital Vernier calipers (Mitutoyo, Model 500-1f16). These calipers are accurate to within 0.01mm. The tendon was then rotated 90 degrees and a second width measure of the callus was taken. Ninety degree reference points were determined by using the proximal, rectangular shaped, fibrocartilaginous end of the tendon specimen as a guide. The tendon callus was measured first with the wider flat surface of the fibrocartilage held flat against the bottom of the petri dish and then measured with the narrow surface held flush with the

Plate 3.5: Optical Comparitor

Plate 3.6: Reflected Tendon Specimen on Optical Comparitor Screen

bottom of the dish.

Following cross sectional area measurement, each tendon was marked with two fine felt pen dots. The dots were marked 2 cm and 4.54 cm from the distal end of the tendon. These two dots served as reference points to ensure the accuracy of the clamping procedure of the tendon. The more distal dot marked the reference point for the lower edge of the upper clamp and the proximal dot marked the reference point for the upper edge of the lower clamp. The 2.54 cm distance between the two dots corresponded to the gauge length of the tendon specimen to be tested. Tendon specimens were then prepared for testing by first attaching a 10 gram weight to the proximal fibrocartilaginous end of the tendon and then placing the distal end of the tendon between a folded, saline soaked, densely woven, burlap material. The distal felt pen dot mark was aligned with the edge of the burlap material (Plate 3.7).

Tensile testing was conducted on an Instron (Model TTD control panel & Model FB load frame) Materials Testing Apparatus that was fitted with a 50kg load cell (Model CM) and a 1 meter extension bar. The Instron Material Testing Apparatus was calibrated to ASTM E-4 standards and is accurate to within ±1%. See Plate 3.8 for a set up of the testing apparatus.

The Instron load frame was modified with a specially designed environmental chamber (1m x 30cm), to ensure a

Plate 3.7: Tendon specimen, 10g weight, Burlap wrap and Felt pen reference points

Plate 3.8: Tensile Testing Apparatus: Set-up

standardized testing environment that replicated internal body conditions (37 degrees centigrade temperature, > 98% relative humidity with physiologic saline). The chamber was constructed out of one inch styrofoam and was firmly attached to the lower mobile bar of the Inston machine. Temperature was regulated by two 100 watt bulbs attached to a Precision Scientific thermometer regulator. A vaporizer (Life, Model 462) that pumped in normal saline, was attached by a two inch diameter hose to the back of the chamber. Relative humidity was monitored by wet and dry bulb thermometers. A small fan was attached to the upper portion of the chamber to ensure air adequate circulation. The two grips and the upper load cell extension bar fit within the chamber and were accessed through a removable plexiglass front. See Plates 3.9 and 3.10 for a set up of the environmental chamber, with and without the grips present.

Specially modified Instron pneumatic grips were used in this study (Plate 3.11). The grips were attached to a pressurized air source and closed with a pre-set 90psi pressure. The metal 4.0 cm x 2.4 cm grips were resurfaced with a high density 1.5 mm thick gasket rubber. A second 2 mm strip of rubber was placed along the outer edge of one surface of both the upper and lower grips. The additional rubber strip had a 1 cm space in the central portion of the grip (Figure 3.6). The additional rubber stripping on the edge of the grip served two purposes. The strip itself

Plate 3.9: Environmental Chamber with Grips

Plate 3.10: Environmental Chamber without Grips

Plate 3.11: Instron Pneumatic Grip

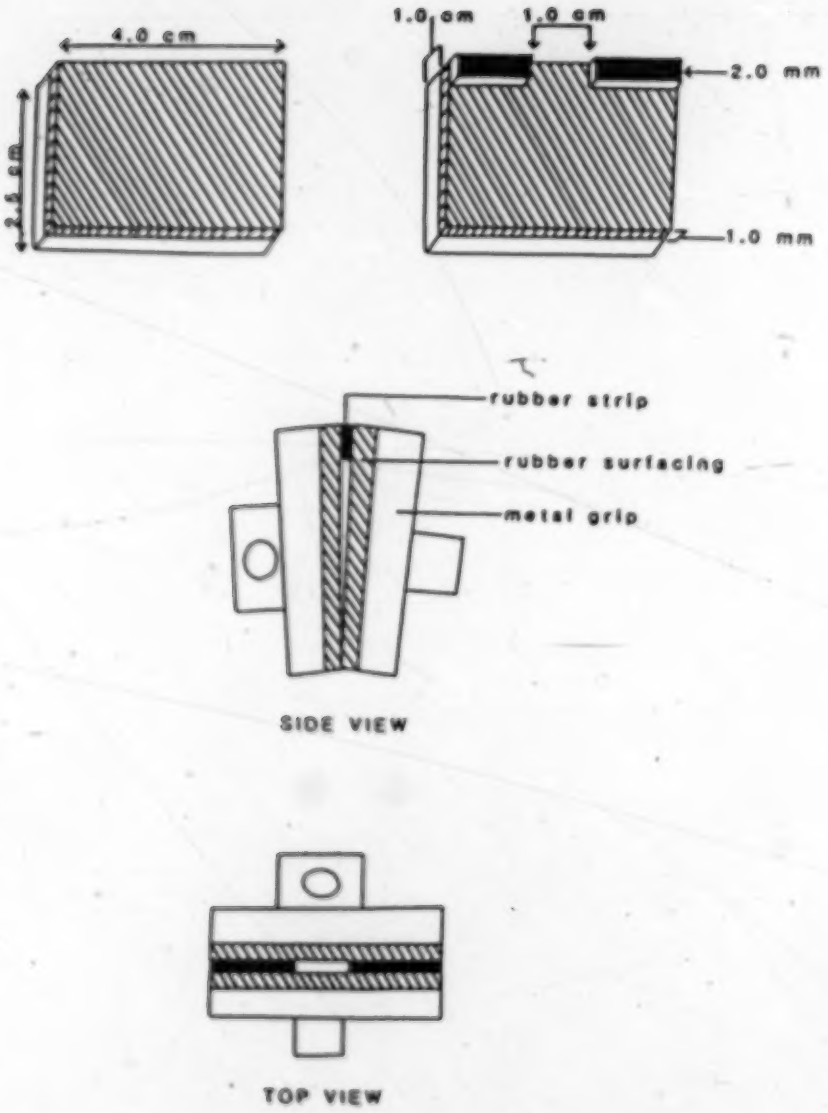

Figure 3.6: Modifications to the Metal Grips.

caused the tendon specimen to be gripped with a graduated pressure wedge-like grip, whereas the central space ensured that there was not a sudden stress concentration on the tendon at the edge of the grips. Grip modifications were done to minimize the risk of tendon failure in, or at, the grips. As well the tendon specimen that was placed between the grips was first placed between two layer of a burlap material. The burlap wrap modification to the gripping procedure ensured that there was no specimen slippage within the grips during testing. With gripping, the soft tendon tissue flowed into the smooth material weave increasing friction between the clamp and tendon specimen.

Prior to testing, the vaporizer and temperature regulator were turned on for fifteen minutes to allow the environmental chamber to reach the desired temperature and relative humidity testing specifications.

At testing, the plexiglass front of the chamber was removed and the wrapped distal end of the tendon specimen was held with fine forceps between the upper clamp grips. The tendon specimen was aligned with the distal dot and edge of the material in line with the central portion of the lower edge of the upper grip. The pneumatic valve was then released causing the tendon to be gripped in the upper grip. The tendon specimen, and 10 gram weight, were then allowed to hang freely between the lower clamp grips. The 10 gram weight acted both as a plumb line to ensure a vertical

orientation of the tendon and as a means of preloading the tendon to a standardized 10 gram load prior to testing.

The gauge length between the grips was then adjusted so that the upper edge of the lower clamp was aligned with the second felt pen dot on the tendon specimen. The more proximal portion of the tendon between the lower grips was then placed between a second piece of folded burlap material and the lower grips were closed. The plexiglass front was then replaced and the tendon specimens was left in the chamber for three minutes to reach an equilibrium with the temperature and relative humidity testing conditions within the environmental chamber.

Tensile testing for all tendons was conducted at an elongation rate of 2.54 cm per minute (100% specimen elongation / minute). A simultaneous load / elongation pen plot was recorded on the Instron strip chart recorder. The chart speed was 25.4 cm per minute and the full scale load was 5 kilograms. Tensile testing continued until the tensile load dropped to within 50 grams of the original baseline. The appropriate tendon identification number was then written on the chart for future identification.

A pilot tensile testing study was also conducted on eight normal chicken Flexor Digitorum Profundus tendons to determine the normal tensile properties of chicken flexor tendons tested under the same strain rate (2.54cm/min; 100%specimen strain/min) and environmental testing conditions

(37° C, 98% R.H.) as the experimental tendons.

Data presentation and analysis

Analysis of the uniaxial tensile testing was based on the load / elongation curve for each tendon. Prior to calculation of the specific material properties, four reference points on the plotted curve were determined (Figure 3.7). The first reference mark was a line extending the initial baseline load below the curve. The second reference point, defined as the point of initial failure, was marked at the first point on the curve that demonstrated a loss in ability of the tendon to resist the applied tensile load. The third and fourth reference points delineated the extent of the linear region of the load / elongation curve. The third point was marked on the curve where the initial convexity towards the baseline changed from curvi-linear to a positively sloped linear orientation. The fourth point, defined as the yield point, was marked at the first deflection away from this linear orientation. Using these four reference points the tensile properties of the tendon specimen (load at initial failure, stress at initial failure, strain to initial failure, material stiffness and energy absorbed to initial failure) could be calculated. The values of each of the tensile properties and the cross sectional area for each tendon specimen were recorded on a raw data form (Appendix II).

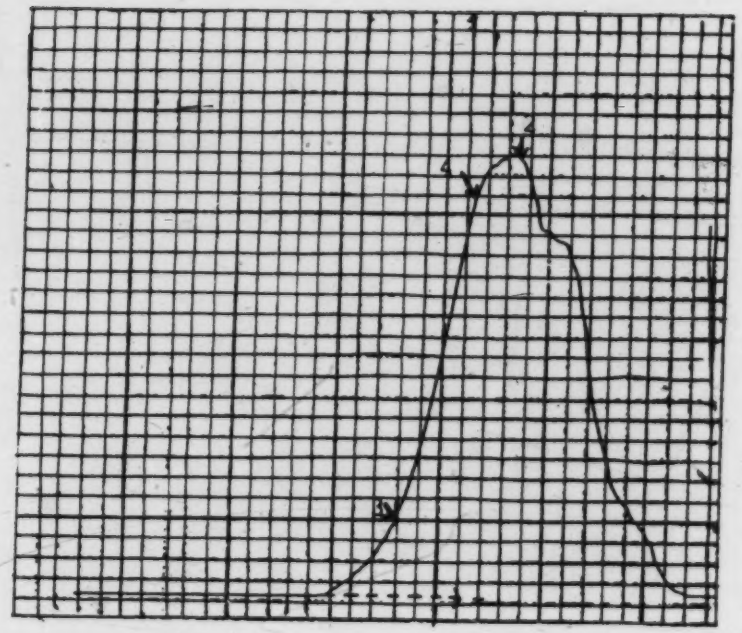

Figure 3.7: Data Analysis Reference Points

1. Baseline extension
2. Point of Initial Failure
3 & 4. Linear Region

Load at initial failure (LIF): LIF was calculated as the distance on the load axis (y-axis), between the baseline load and load at the point of initial failure (Figure 3.8). The y-axis full scale was a 5 kg load, with each of the small squares on the graph equivalent to a 50 gram load.

Stress at initial failure (SeIF): SeIF was calculated by dividing the load at initial failure value by the cross sectional area measure for the tendon callus. Cross sectional area calculations were based on the assumption that the tendon callus was ovoid in shape. Specifically, the calculation involved adding the two diameter measures of the tendon callus and dividing by four to get an average radius measure for the tendon callus. Then using the formula for area of a circle (r^2), the cross sectional area measure was calculated.

Strain at initial failure (SaIF): A direct measure of the tendon specimen strain was not used in this study. In order to make a meaningful comparison of strain measures between tendon specimens, a standardized point for estimation of initial strain was established. By using the two reference points (3 and 4) that delineated the linear portion of the curve, a line of best fit was determined and extended back to intersect with the baseline x-axis. SaIF was then determined in two ways (Figure 3.8):

1. By calculating the distance between the point of intersection of the line of best fit with the x-axis

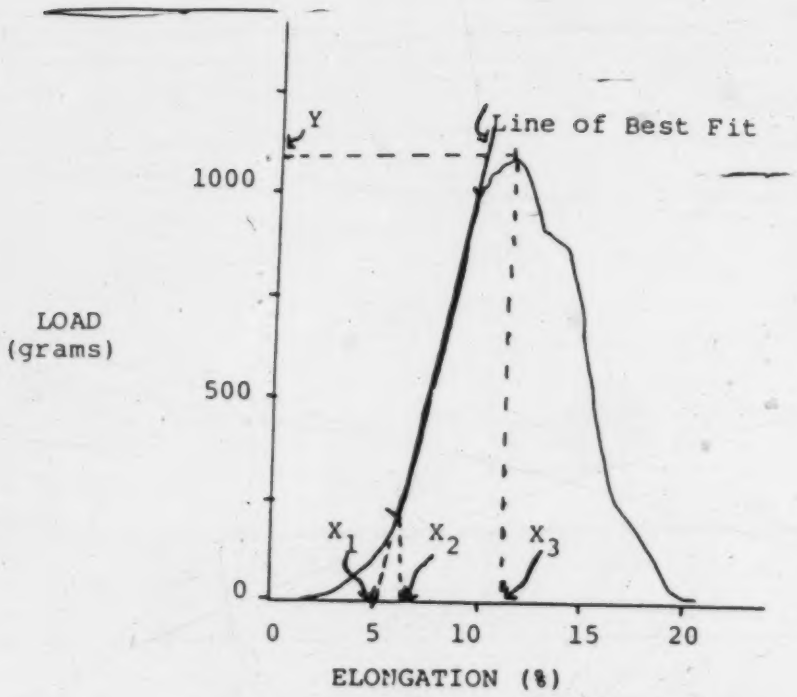

Figure 3.8: Tensile Properties Calculation

Y - Load at Initial Failure

X_1 - Initial Strain - Line of Best Fit

X_2 - Initial Strain - Point of Deflection

X_3 - Strain at Initial Failure

and the point of strain at initial failure (cf. Elliott, 1967). This is referred to as strain-a.

2. By calculating the distance between the x-co-ordinate of the point of deflection of the line of best fit away from the load / elongation curve and the point of strain at initial failure (cf. Viidik, 1973). This is referred to as strain-b.

Distance along the x-axis is proportional to the distance between the testing apparatus grips, as long as the test specimen does not slip in the grips during testing. The ratio of chart speed (10"/min; 25.4 cm/min) to specimen strain rate (1"/min, 2.54 cm/min), was 10:1. Therefore, a distance along the x-axis of 1" (2.54cm) was equivalent to 10% specimen strain, with each of the small squares on the graph equal to 1% specimen strain.

Material stiffness (MS): The MS of the specimen was determined by dividing stress at initial failure by strain to initial failure (Wainwright, Biggs, Currey & Gosline, 1976, pp 9). Separate MS measures, utilizing both strain-a and strain-b calculations, were determined for each tendon.

Energy absorbed to initial failure (EAIF): EAIF to initial failure was calculated by determining the area under the curve from the point of initial strain-a, to strain at initial failure. In this study energy absorbed was determined for the whole specimen and not per unit volume, as

differential processes of energy absorption throughout the whole test specimen (normal tendon vs healing tendon) were not able to be factored out with calculations.

A Hipad^{TM} digitizer (Houston Instruments, Model DT11AA) was used to make this calculation. The mean value of two digitized values for each curve was used for this measure. EAIF, in Joules (Newtons x Meters), was then calculated with the known conversion ratio of 1-10 x 10 square (.5 kg x 2.54cm) on the graph paper = to .125 joules / a digitized value of 598.35 mm^2.

Data Analysis: Analysis of the data was done on the University of Alberta mainframe computer, utilizing a userproc uanova subprogram of the SPSSX statistical software program.

A 2 x 5 (treatment condition x healing period) analysis of variance, with one within subject factor (treatment condition; controlled passive motion vs immobilization) and one between subject factor (healing period; 10, 15, 20, 25 and 30 days) was run for: (1) each of the tensile properties examined (LIF, SeIF, SaIF, MS and EAIF) and, (2) for the cross sectional area measurement of the tendon callus. Significant main effects for treatment condition, healing period and interaction effects between treatment condition and healing period were examined for each of these dependent variables.

Post hoc analysis for significant healing effects was

conducted with an a priori planned orthogonal analysis between days 15 and 25, and days 20 and 25. These two healing period intervals were considered, a priori, to be of importance when it came to delineating a time period during which significant healing effects could have clinical significance. Significance levels for each analysis of variance and planned orthogonal posthoc analysis were set at alpha equal to or less than .05.

Posthoc analysis for simple main effects of controlled passive motion vs immobilization, at each of the five healing periods was examined for any significant main treatment effect found. Significance level for this analysis was set at alpha equal to or less than .01 (.05/5) to avoid the greater probability of making a type 1 statistical error with this posthoc multiple comparison technique.

Chapter IV

RESULTS AND DISCUSSION

Experimental animal

Thirty mature, white leghorn roosters were utilized in this study conducted to compare the effects of early postoperative management regimes on the early tensile properties of healing chicken flexor tendons.

Chickens were purchased from a local supplier (FM Farms Ltd.; Rochester, Alberta) and were all selected from the same breeding stock. At the time of surgery the roosters were between the ages of 27 to 31 weeks (mean age = 28.77 weeks) and weighed between 1730 to 2275 grams (mean weight = 2003.16 grams) with suspension jackets, casts and splints (approx. weight of jacket, casts and splints = 300 grams).

Nine chickens were excluded from the study and replaced. No chickens were excluded due to reasons attributable to the experimental manipulation under investigation. Reasons for exclusion and replacement were:

1. Four of the initial fifteen chickens died within three days of purchase from an illness which caused severe diarrhea and respiratory distress. Another chicken from this initial group was sent for necrotopsy to investigate the cause of the illness. Although no specific cause for the illness was found,

all of the remaining chickens from the initial shipment utilized in the study, and all future chickens purchased, were placed on prophylactic antibiotics (Nitrofurazone Powder, NF). The antibiotic was administered in the drinking water and commenced on the seventh preoperative day and continued until sacrifice.

2. One chicken died at the 18th postoperative day due to strangulation on a lace of the suspension jacket.
3. One chicken was excluded preoperatively due to a crush injury of the middle toe sustained during cage cleaning.
4. Two chickens were excluded from the study at the time of surgery because of concern over a faulty suture. The suture easily attenuated during the repair and frayed and broke when the knots were tied. Instead of repeating the repair and causing further surgical trauma to the tendon, extra knots were tied and these chickens were followed postoperatively until the 10th and 15th postoperative day healing periods. The tendons were dissected out, fresh frozen and set aside for possible histological study or reliability testing of the cross sectional area measure.

Surgical procedure and postoperative management

All chickens tolerated well the 45 minute surgical

procedure conducted under local anesthetic, and were placed back up in suspension in their individual cages within 1/2 to 1 hour following surgery. No chicken appeared to be in any postoperative discomfort or developed any signs of acute illness. However, all chickens did remain quite lethargic during the first 24 to 36 postoperative hours and slept most of the day. Following this initial quiet period the chickens began to spend more time awake each day and in most instances began to eat, drink and preen themselves normally within the first four to seven days.

A small number of the chickens (5 out of 30; 16.6%) that not tolerate the suspension system. These chickens only occasionally ate or drank and had to be maintained on supplemental feeding and watering throughout the total postoperative treatment period. These same chickens were also more restless in the suspension system and made frequent attempts to free themselves.

Reasons for this poor tolerance in some chickens were unclear, although it was noted that these same chickens were also more restless during the pre-operative acclimatization period and may simply have been more restless chickens due to organismic variables. Another reason for this postoperative restlessness may be that the suspension jackets did not fit properly and were uncomfortable. At the time of sacrifice, the restless chickens were noted to have small friction rubs along the inner thigh and posterior axillary regions. These

areas may have been the source of discomfort, or may have been caused by the chickens flapping and kicking with attempts to free themselves. None of the thirty experimental chickens had any pressure area along a bony prominence or weight bearing area within the jacket.

Perhaps poor tolerance to the suspension system could be avoided in future studies by:

1. Excluding from surgery the few chickens that do not settle with pre-operative acclimatization.
2. Re-designing the suspension jacket to provide for a more customized fit for each individual chicken.

Initial dressing removal

At the time of initial dressing removal it was noted that no incision had developed any signs of infection. All incisions were well approximated and clean. With some dressing removals there was some fresh bleeding from the incision. However, with light compression all evidence of bleeding was quickly stopped. In approximately 25% of the incisions there was also some mild subcutaneous bruising along the incision. However, there was no evidence in any of the incisions of any significant amount of postoperative bleeding. All of the feet demonstrated bruising in the medial webspace and around the base of the metatarsal. These two sites correspond to the location of the local anesthetic injection and tourniquet placement.

Controlled passive motion regime

All chickens tolerated the early controlled passive motion toe manipulations without any signs of discomfort. The chickens had access to food during this time and in most instances, after the first couple of treatments, ate throughout the treatment session.

X-ray excursion study

Reliability of the results of the x-ray excursion study is questionable, as there was no standardized positioning at the time of x-ray. Therefore, differences in wire position between x-ray views may be in part due to different x-ray angles. However, passive motion of the flexor tendons relative to the axis of the head of the second phalanx was consistently demonstrated with the passive joint manipulations in all three chickens studied in the x-ray excursion study. With consideration of the unknown reliability of this measurement, there was an average overall excursion of:

1. 4 mm for both tendons with composite joint flexion.
2. 1.5 mm of maximal differential excursion between the FDP and FDS tendons with combined DIP and MIP passive flexion (Plates 4.1 and 4.2).
3. 1 mm for either tendon relative to the MIP joint with any of the isolated joint motions studied.

Plate 4.1: X-ray of Imbedded Wires: Position of Immobilization

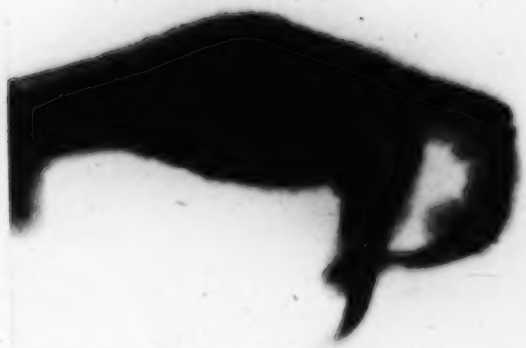

Plate 4.2: X-Ray of Imbedded Wires: Position of DIP and MIP Flexion

Consistency of flexor tendon excursion demonstrated in the x-ray excursion study, was felt to be sufficient evidence in support of the validity of an early controlled passive motion regime in producing passive flexor tendon excursion in the chicken toe at the level of repair.

Dissection

At the time of dissection all tendons were found to be clinically intact. Clinical intactness was defined as clearly approximated tendon ends, with a gap of no greater than 1.5 mm between the two tendon ends. Eight of the sixty tendons (13.3%) dissected, were defined as clinically intact but having poor clinical status. See Table 1. for a summary of the poor status tendons.

The eight poor status tendons were spread between the five healing periods, with three out of the eight tendons (37.5%) from the immobilized treatment group and five out of the eight tendons (62.5%) from the controlled passive motion treatment group. Four of the eight tendons (50%) were not completely approximated, they had a gap of less than 1.5 mm with the suture still in both tendon ends. Two of the eight tendons (25%), had a gap of less than 1.5 mm with the suture found only in the proximal tendon stump. Two of the eight tendons (25%) were found to be closely approximated, but the tendon callus was poorly defined, soft and very vascular.

One tendon (immobilized treatment group / 10 day healing

Table 1.

Poor Clinical Status Tendons at Time of Dissection

Chicken Number	Healing Period	Treatment Condition	Repair Status	Suture Status
27	4	Immobilized	attenuated (<1.5mm gap)	Suture intact
11	1	CPM	"	"
30	3	CPM	"	"
30	3	Immobilized	"	"
3	5	CPM	"	Suture in distal stump
16	3	Immobilized	"	"
17	4	CPM	approximated (soft, vascular callus)	Suture intact
22	5	CPM	"	"

period), in a chicken already excluded and replaced in the study due to concern of faulty suture, was found to be no longer clinically intact at the time of dissection. The tendon had retracted proximally leaving a gap of approximately 2 centimeters between the cut tendon ends. There was a large hematoma in the gap at the time of dissection. Follow-up investigation of the tendon ends showed that the suture had failed at the site of knotting and pulled out of the proximal tendon stump (Plates 4.3 and 4.4). This non-experimental tendon was the only clinical rupture in the study, all of the experimental tendons classified as having poor clinical status at the time of dissection met the requirement of being clinically intact and were, therefore, included in the tensile testing procedures.

The assumption that the incised tendon sheath edges would be approximated well enough with skin closure to allow for adequate healing appears to have been appropriate. All flexor tendon sheaths at the time of dissection were found to be healed at the site of sheath incision. Although it was impossible to tell without histological study, if the sheath healed by a connective tissue scar or proliferation of synovial tissue, there was no doubt that there was always a defined barrier of tissue across the sheath incision site that separated the underlying tendons from the overlying subcutaneous connective tissue.

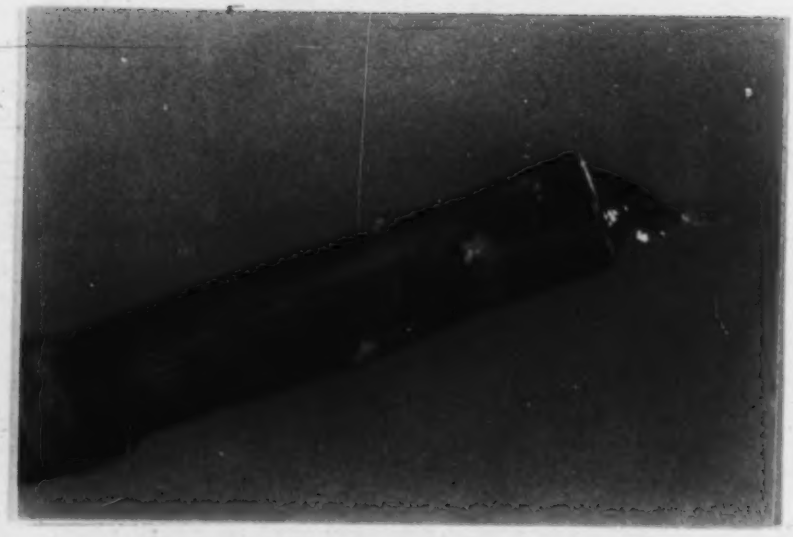

Plate 4.3: Ruptured Tendon: Proximal Stump

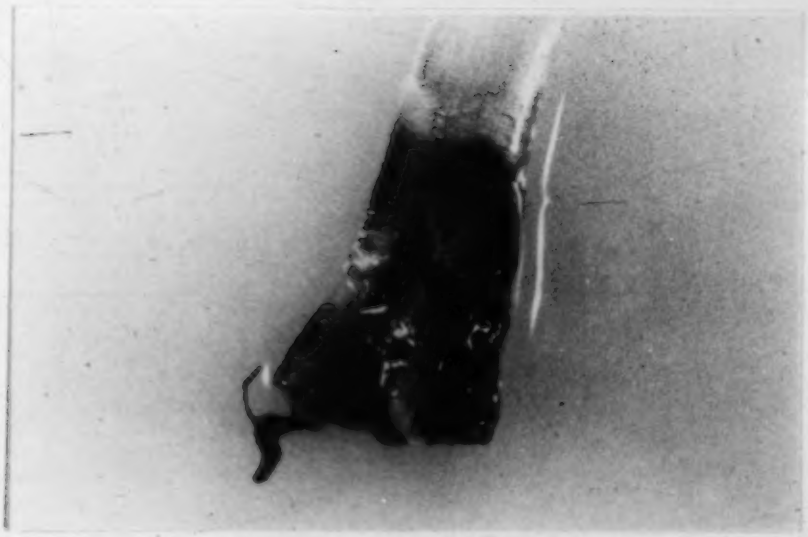

Plate 4.4: Ruptured Tendon: Distal Stump and Failed Suture

Tensile testing

All experimental tendons failed at the repair site with tensile testing. Following testing, the tendons were removed from the grips and examined to ensure that the dots were aligned with the edge of the gripped regions to check the accuracy of the gripping procedure. As well, imprints of the material weave in the tendon were examined for evidence of shredding or slippage of the tendon specimen within the grip. All tendons were gripped accurately to within 1 mm of the reference dots, and no tendon demonstrated any evidence of failure or slippage in the grip. Therefore, no experimental tendon specimens were excluded from the study due to technical difficulties with tensile testing or gripping procedures.

A summary of the tensile properties of the normal FDP tendons tested is presented in Table 2. None of the normal tendon specimens tested demonstrated any evidence of slippage in the grips. However, of the eight normal tendon specimens tested, four failed at the upper grip and were excluded from analysis.

Consistency of values for the material properties examined for the four tendons that did fail between the grips, suggests that this small sample size was adequate to determine baseline normal values for chicken FDP tendons tested under the same experimental conditions as the experimental healing FDP tendons.

Table 2.

Tensile Properties for Normal Flexor Digitorum Profundus Tendons (Pilot Study)

Chicken #	Cross-Sectional Area (mm^2)	Load at Failure (kg)	Stress at Failure (kg/mm^2)	Strain to Failure (%)	Material Stiffness (kg/mm^2)	Energy Absorbed to Failure (Joules)
1	1.91	29.50	15.44	25.50	60.55	.606
2	1.96	30.00	15.31	24.25	63.13	.627
3	2.01	31.50	15.67	24.50	63.96	.640
4	1.89	28.75	15.24	25.75	59.18	.592
Mean	1.94	29.94	15.42	25.04	61.71	.616

Data analysis

The data analysis was conducted on the results of all sixty experimental tendons examined in the study. No tendons were excluded, as all tendons were found to be clinically intact at the time of dissection and no tendons were excluded due to technical difficulties with tensile testing.

A. Inter-rater reliability

The inter-rater reliability coefficients, as determined by an Inter-class Correlation Coefficient (ICC) for Load at Initial Failure (LIF), Cross-sectional area, Strain to Initial Failure (SaIF) and Energy Absorbed to Initial Failure (EAIF) measures, were all equal to .9 (Bartko & Carpenter, 1976). See Appendix I. for a summary of the ICC calculations.

B. Analysis of variance

Significant differences of $p \leq .05$ found with the 2 x 5 (treatment condition x healing period) analysis of variance for each dependent variable examined (LIF, SeIF, SaIF, MS, EAIF and Cross-sectional area) were:

1. Treatment effects (X): (H_0: $x_1 = x_2$)

(i) Load at Initial Failure (LIF)

[$F(1,25) = 9.31$, $p = .005$].

(ii) Stress at initial failure (SeIF)

[F(1,25) = 5.55, p = .03].

(iii) Energy absorbed to initial failure (EAIF)

[F(1,25) = 5.56, p = .02].

2. Healing effects (Y): (H0: Y1= ... Y5)

(i) Stress at initial failure (SeIF)

[F(4,25) = 3.64, p = .02]

(ii) Strain a and b to initial failure (SaIF)

[F(4,25) = 4.22 and 3.41, p = .01 and .02 respectively].

(iii) Material stiffness a and b (MS)

[F(4,25) = 6.73 and 6.33, p = .001 and .001 respectively].

3. Interaction effects (XxY): (H0: X1Y1= ... X2Y5)

No significant interaction effects were found.

Table 3 summarizes all the F-ratios and probability values for treatment, healing and interaction effects for each dependent variable examined with analysis of variance. Table 4 summarizes all dependent variable treatment condition x healing period cell means. Figures 4.1 and 4.2 graphically depicts these cell means for significant ANOVA treatment condition and healing period effects.

C. Planned orthogonal posthoc analysis

Significant cell mean differences of $p \leq .05$ found with a planned orthogonal posthoc analysis between days 15 and 25

Table 3.

Cell Mean Values for each Dependent Variable

Variable/condition	Healing Period				
	10 days	15 days	20 days	25 days	30 days
Load (grams)					
Immob	671.7	859.2	760.0	883.3	1157.5
CPM	795.0	1036.7	1086.7	1541.7	1518.3
Stress(g/mm^2)					
Immob	70.1	69.2	76.2	87.4	89.0
CPM	81.5	76.6	81.7	136.7	130.2
Strain - a(%)					
Immob	5.8	6.0	6.7	4.7	4.3
CPM	7.8	5.3	5.7	4.9	5.0
Strain - b(%)					
Immob	4.9	5.2	5.7	3.9	3.7
CPM	6.7	4.4	4.9	4.4	4.3
Stiffness - a (g/mm^2)					
Immob	1247.6	1329.1	1199.8	1990.6	2083.4
CPM	961.2	1478.5	1473.1	2891.4	2598.0
Stiffness - b (g/mm^2)					
Immob	1502.7	1550.3	1417.8	2396.1	2310.3
CPM	970.5	1802.8	1757.5	3374.4	3108.9
Energy (Joules)					
Immob	.061	.068	.081	.052	.073
CPM	.089	.078	.094	.108	.116
Cross Section (mm^2)					
Immob	10.0	11.4	9.4	10.7	12.1
CPM	9.6	14.1	13.6	11.3	11.8

Table 4.

Analysis of Variance F-ratios and Probability Values for Treatment, Healing and Interaction Effects

Variable	Treatment Effect	Healing Effect	Interaction Effect
Load			
F-ratio	9.31	1.82	.75
Prob.	.005(*)	.15	.57
Stress			
F-ratio	5.55	3.64	.90
Prob.	.03(*)	.02(*)	.48
Strain - a			
F-ratio	.36	4.22	1.46
Prob.	.55	.01(*)	.24
Strain - b			
F-ratio	.38	3.41	1.51
Prob.	.54	.02(*)	.23
Stiffness - a			
F-ratio	2.26	6.73	.91
Prob.	.14	.001(*)	.47
Stiffness - b			
F-ratio	2.05	6.33	1.05
Prob.	.16	.001(*)	.40
Energy			
F-ratio	5.56	.26	.47
Prob.	.02(*)	.89	.75
Cross Section			
F-ratio	2.46	.81	1.07
Prob.	.13	.53	.39

Note: * $p \leq .05$

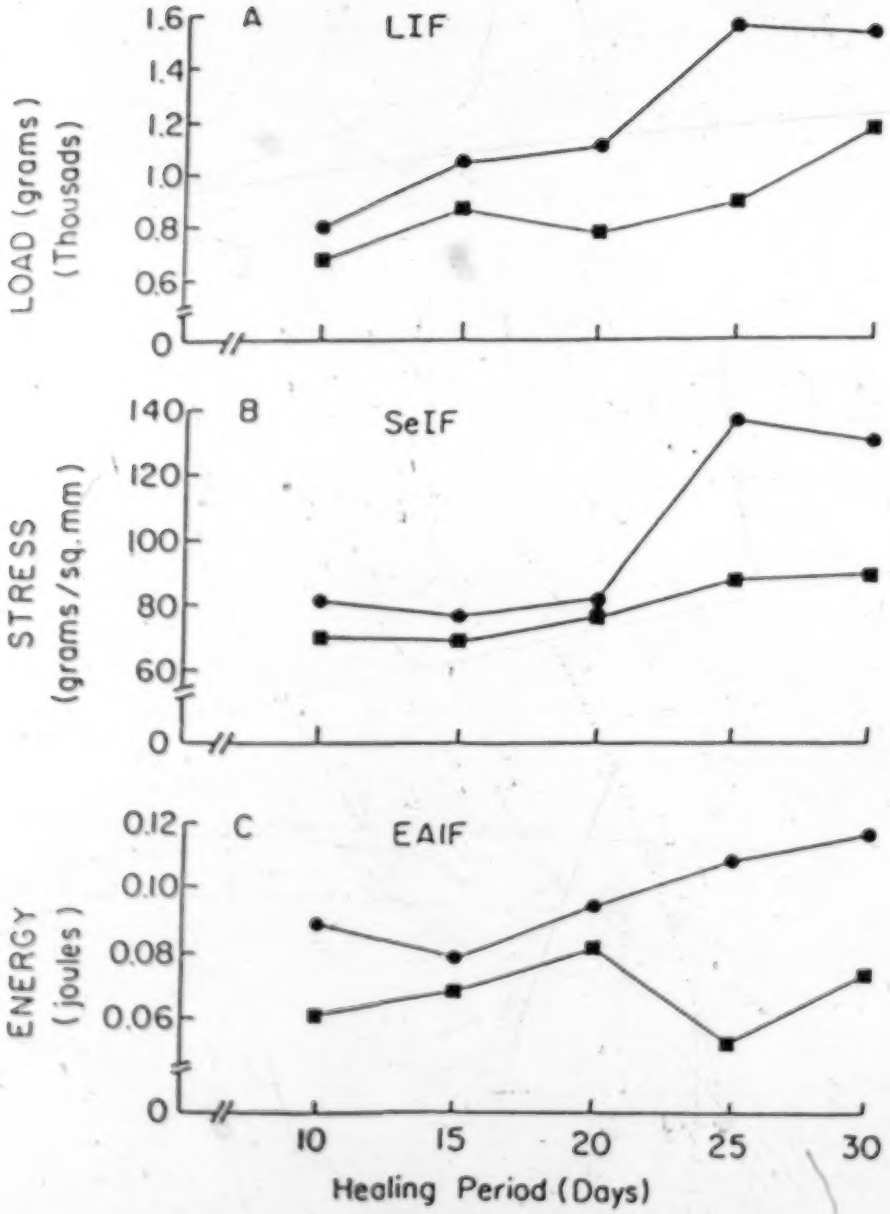

Figure 4.1: Cell Means for Significant ANOVA Treatment Condition Effects: (●) CPM vs (■) Immob

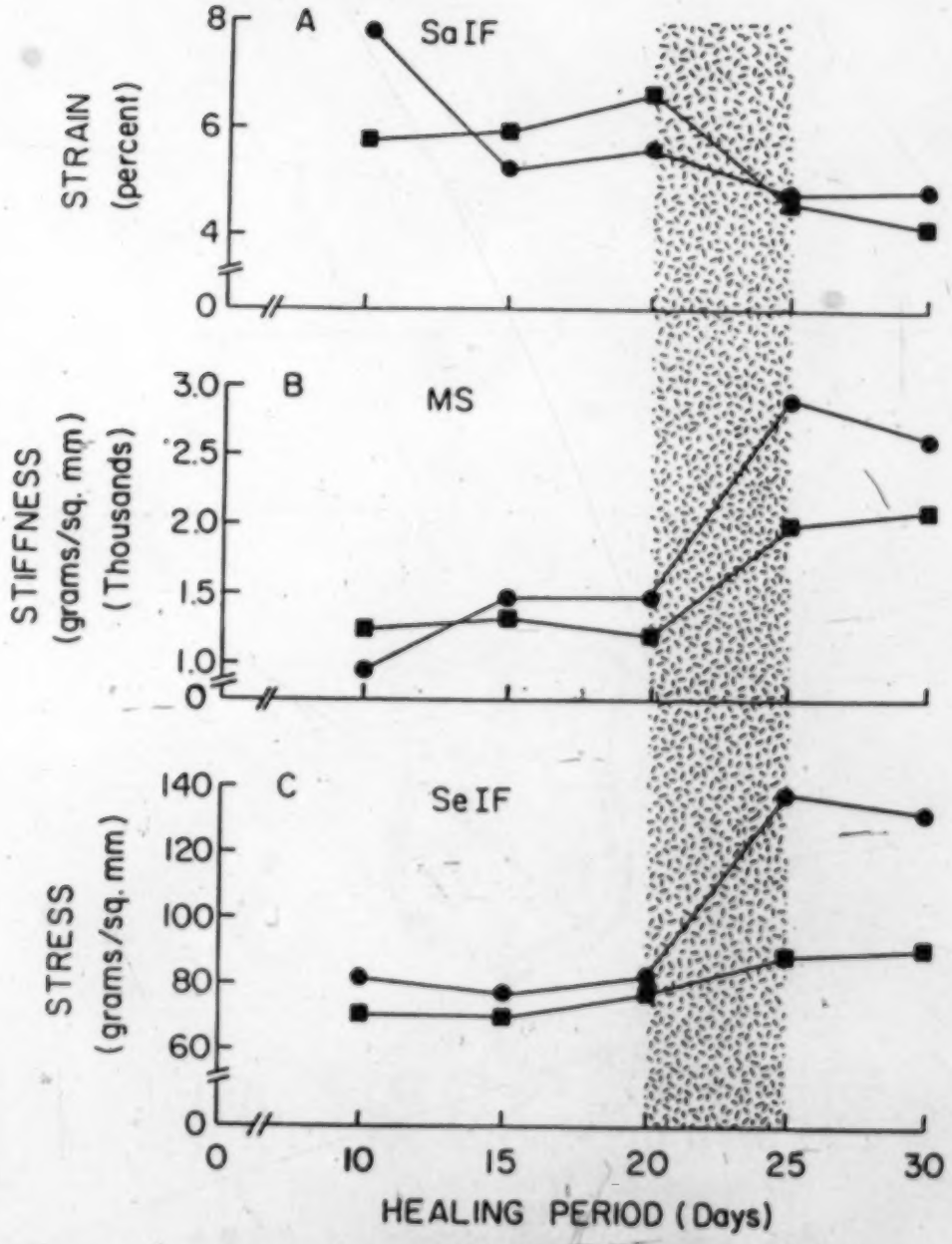

Figure 4.2: Cell Means for Significant ANOVA Healing Period Effects:
(•) CPM vs (▪) Immob

and days 20 and 25 for significant healing effects found with analysis of variance were:

1. 15 to 25 day healing period:

(i) Stress at initial failure (SeIF)
[$F(1,25) = 1.09$, $p = .02$].

(ii) Material stiffness a and b (MS)
[$F91,25) = 9.45$ and 8.64, $p = .005$ and .007 respectively].

2. 20 to 25 day healing period:

(i) Stress at initial failure (SeIF)
[$F(1,25) = 5.36$, $p = .03$],

(ii) Strain a and b at initial failure (SaIF)
[$F(1,25) = 4.86$ and 4.08, $p = .04$ and .05 respectively].

(iii) Material stiffness a and b (MS)
[$F(1,25) = 10,72$ and 9.96, $p = .003$ and .004 respectively].

Table 5. summarizes all the F-ratios and probability values for the planned orthogonal analysis for each of the significant healing effects found with analysis of variance.

D. Simple main effects posthoc analysis

Significant cell differences of $p \leq .01$ between the controlled passive motion treatment group and the immobilized

Table 5.

F-ratio and Probability Values for the Planned Orthogonal Posthoc Analysis of Significant Healing Effects

Variable	15 to 25 day Healing Period	20 to 25 day Healing Period
Stress		
F-ratio	7.49	5.36
Prob.	.01(*)	.03(*)
Strain - a		
F-ratio	1.72	4.86
Prob.	.20	.04(*)
Strain - b		
F-ratio	1.17	4.08
Prob.	.29	.05(*)
Stiffness - a		
F-ratio	9.45	10.72
Prob.	.005(*)	.003(*)
Stiffness - b		
F-ratio	8.64	9.96
Prob.	.007(*)	.004(*)

Note: * $p \leq .05$

treatment group at the different healing periods examined were:

1. 10 day healing period

No significant cell differences

2. 15 day healing period

No significant cell differences

3. 20 day healing period

No significant cell differences

4. 25 day healing period

(i) Load at Initial Failure (LIF)

[$F(1,25) = 6.72$, $p = .01$]

5. 30 day healing period

No significant cell differences

Table 6 summarizes the F-ratios and probability values for the simple main effects posthoc analysis at the 25 day healing period for each of the significant treatment effects found with analysis of variance.

Table 6.

F-ratio and Probability Values for Simple Main Effects Posthoc Analysis for Treatment Effects (25 day Healing Period)

Variable	25 day Healing Period
Load	
F-ratio	6.72
Prob.	.01(*)
Stress	
F-ratio	5.09
Prob.	.03
Energy	
F-ratio	3.86
Prob.	.06

Note: * $p \leq .01$

Null hypotheses discussion

Results of the data analysis will be discussed within the framework of the specific research null hypotheses of the study.

A. Treatment effect null hypothesis:

> That the early controlled passive motion tendon treatment group would not have significantly different values for any of the tensile properties examined when compared to the early postoperative immobilization tendon treatment group.

Results of this study allowed for rejection of the treatment effect null hypothesis for Load at Initial Failure (LIF), Stress at Initial Failure (SeIf) and Energy Absorbed to Initial Failure (EAIF).

Findings of a significantly greater LIF for the controlled passive motion treatment group as compared to the immobilized treatment group, throughout the early postoperative 20 day healing period (10 days to 30 days) examined in this study, is consistent with the reported findings of other authors (Gelberman et al, 1982; Hitchcock et al, 1987).

A consist finding in independent studies, of an increased rupture load with early postoperative controlled mobilization of flexor tendons as compared to immobilized tendons, is strongly supportive of the validity of this experimental finding. Experimental evidence of increased rupture load in healing tendons managed with early controlled tendon mobilization suggests that flexor tendons managed clinically with an early postoperative controlled tendon mobilization

program, will also have a significantly greater tolerance to tensile forces throughout the early fibroplastic phase (5 to 21 days) of healing and continue to demonstrate this increased tolerance into the remodelling and maturation (> 21 days) phase of tendon healing.

When comparing rupture load (LIF) between two early postoperative treatment groups, it is not possible to differentiate between differences in strength due to true material differences within the tendon, or differences in strength due to differences in tendon bulk. Differences in bulk, or cross sectional area, may then account for part, or all, of the differences in rupture strength found between the two treatment groups. The absence of a significant difference in cross sectional area between the two treatment groups in this study suggests, however, that this is not the case.

Comparing SeIf between the two different treatment groups in this study, allowed for a direct comparison of the true material strength of the healing tendons, independent of the size of the tendon specimen being examined. The finding of a significantly greater SeIf throughout the early 20 day postoperative healing period examined, for controlled passive motion tendons as compared to immobilized tendons, demonstrates a greater true material strength of the healing flexor tendons managed with early controlled passive motion. This finding suggests not only that tendons managed

clinically with early controlled passive motion will demonstrate a greater tolerance to tensile forces when compared to immobilized tendons, but also that this increased tolerance may be due to actual morphological differences within the healing tendon. To date, there have been no other studies published that have demonstrated this finding of an increased material strength in healing flexor tendons managed with early controlled passive motion as compared to immobilized tendons.

The present study did not directly examine the morphology of the two different tendon treatment groups studied. Therefore, any correlations between morphological differences and differences in material strength are only speculative. However, some research supports, in theory, a possible relationship between an enhanced material strength and morphological differences between the two early postoperative treatment groups. These morphological differences may be:

1. An enhanced intrinsic fibroblast proliferation within the tendon (Gelberman et al, 1985, Nelson et al, 1985).
2. Orientation of fibroblasts and collagen along the lines of tensile stresses generated with early postoperative controlled passive motion (Thorngate, 1958; Bassett, 1971; Becker & Diegelmann, 1984).

Findings from this study also demonstrated a significantly

greater EAIF for early controlled passive motion tendons as compared to immobilized tendons throughout the 20 day healing period. Examination of the EAIF at the various healing periods demonstrates that, following a slight drop in the EAIF between the 10 to 15 day healing period, the controlled passive motion tendons show a consistent rise in EAIF throughout the remainder of the healing period. Conversely, the immobilized tendons show a drop in EAIF at the 20 day healing period. This differential finding for EAIF at the 20 day healing period, may be accounted for by the concomitant finding of a significantly greater rupture load (LIF) for the controlled passive motion treatment group as compared to the immobilized treatment group, at the 25th postoperative day.

Area under the tensile curve (EAIF) is dependent upon rupture strength (LIF), deformation at failure (SaIF) and the relationship of rate of development of deformation to the rate of development of load bearing capabilities (MS), in the test specimen. This study demonstrated an inverse relationship between development of MS and SaIF measurements in the healing flexor tendons, in which the MS increased and the SaIF decreased. Due to this inverse relationship between MS and SaIF, area under the load / elongation curve would be expected to decrease as the healing period progresses. However, if there was also a concomitant increase in the strength of the tendon during this same period, as was

demonstrated with LIF at the 25th day with the early controlled passive motion treatment group, the area under the tensile curve would be expected to increase.

A possible explanation for the differential findings for EAIF and LIF between the two treatment groups at the 20 to 25 day healing period, may be related to differential processes of remodelling and crosslink maturation of connective tissue wounds at this point in time. As discussed by Madden and Peacock (1971), the attributable rise in tensile strength of connective tissue wounds at this three week healing period is related to a complex mechanism of collagen remodelling and crosslink maturation within the wound. If with early controlled passive motion, the fibroblast proliferation and collagen production within the tendon callus is influenced by tensile stresses to be laid down in a parallel fashion in relation to the lines of stress, then the early controlled passive motion tendons would conceivably:

1. Not have to go through the process of secondary remodelling into parallel tendon like tissue and, therefore, would progress directly into crosslink maturation processes.
2. Demonstrate a more efficient crosslink maturation process, through the development of an orderly pattern of inter- and intra-fiber crosslinks within an already established parallel aggregation of collagen fibers in the healing callus.

B. Healing effect null hypothesis:

> That tendons in both the early postoperative immobilization and the early controlled passive motion treatment groups would show no significant change across the five healing periods examined in any of the material tensile properties examined.

Results from the analysis of variance allowed for rejection of the healing effect null hypothesis for Stress at Initial Failure (SeIF), Strain to Initial Failure (SaIf) and Material Stiffness (MS).

Results of the analysis for SaIf and MS, as previously stated, show an inverse relationship between SaIf and MS. Across the 20 day healing period examined in this study, the SaIF for both treatment groups decreased significantly, whereas, MS increased significantly. This finding of an inverse relationship between MS and SaIF, as well as, a significant increase in material strength (SeIF) across the 20 day healing period, is consistent with what would be expected to happen to these material properties if temporal changes in the healing tendon tissue composition are taken into consideration.

Composition of the healing tendon changes from a predominantly viscous material composed of a blood clot and inflammatory exudate during the inflammatory phase of healing (5 to 7 days), to a more fibrous tissue composed primarily of collagen fibers and their supporting fibroblasts during the fibroplastic phase of healing (7 to 21 days) (Potenza, 1962; Lindsay, 1964). As viscous elements decrease in the tissue

matrix the SaIF would also be expected to decrease. Whereas as the fibrous content increases in the tissue matrix the MS and SeIF would be expected to increase (Viidik, 1973).

Delineation in this study, of a significant change in the material properties SeIF, SaIF and MS, between days 20 and 25 is also consistent with the reported findings in the literature. A rise in rupture load of healing tendons between the 19th to 21st postoperative days was first reported by Mason and Allen (1941), and as described by Madden and Peacock (1971), correlates with the remodelling and crosslink maturation processes of connective tissue healing at this same period in time.

C. Interaction effect null hypothesis:

> That the early controlled passive motion tendon treatment group would not have a significantly different rate of change in any of the tensile properties examined as compared to the early postoperative immobilization treatment group.

Results of the data analysis did not allow for rejection of the interaction null hypothesis for any of the dependent variables examined. This suggests that early controlled passive tendon mobilization regimes do not significantly increase, or decrease, the rate of healing of flexor tendons. Therefore, the assumption that early controlled passive motion tendons heal more slowly than immobilized tendons was not substantiated by the findings of this study.

This study demonstrates that early postoperative controlled passive motion tendons have both an improved material tensile strength and a similar rate of healing, compared to early postoperative immobilized tendons. Therefore, a reasonable conclusion may be, that if it is accepted clinically safe to institute a protected active tendon mobilization program following three weeks of early postoperative immobilization, it should be equally safe to commence a protected active tendon mobilization program at this same time period following an early postoperative controlled passive motion regime.

However, there is a substantial difference in magnitude of the tensile properties examined, between the healing experimental FDP tendons at the end of the 30 day healing period and the normal FDP tendons examined in the pilot study. See Table 7 for a comparison of these tensile property values.

Based on the large discrepancy found in this study, between tensile properties values for normal FDP tendons and healing FDP tendons at thirty days, protection of the healing flexor tendon against excessive stresses and strains during the early stages of active tendon mobilization should still be advocated. It would be advisable, during the early stages of active tendon mobilization for the clinician to:

1. Proceed cautiously with graduation of the active tendon mobilization programs.

Table 7.

% of Normal Tensile Property Values at the 30 Day Healing Period

Variable/Condition	% Normal
Load	
Immob	4
CPM	5
Stress	
Immob	.5
CPM	.8
Strain	
Immob	17
CPM	20
Stiffness	
Immob	4
CPM	5
Energy	
Immob	12
CPM	19

2. Continue with some means of protective splinting to guard the healing tendon against excessive and unexpected strains.

Chapter V

SUMMARY, CONCLUSIONS AND CLINICAL IMPLICATIONS

Summary

After primary flexor tendon repair in the fibro-osseous tunnel, active tendon mobilization is usually commenced at three weeks following an early postoperative immobilization treatment regime. In contrast, when early postoperative controlled passive motion regimes are utilized, initiation of active motion has not been advocated until usually four and one half weeks and even up to ten weeks postoperatively.

The reasons for this delay in active tendon motion with early controlled passive motion are unclear. Caution, for fear of rupture, has been advocated on the assumption that tendons managed by controlled passive motion heal more slowly, and so develop tensile strength more slowly than tendons managed by early postoperative immobilization. Reported findings for the importance of motion for synovial fluid diffusion within tendon, and the beneficial effect of stress on healing connective tissues, suggests that the opposite may be true. Tendons managed with controlled passive motion regimes during the early postoperative healing period have the potential to heal with a more efficient early healing process due to improved synovial fluid nutrition, and to develop morphological changes that enhance development of

early functional tensile material properties due to the beneficial effects of stress on healing connective tissues.

The present study was conducted to compare the early postoperative tensile properties of surgically divided an repaired chicken Flexor Digitorum Profundus tendons, between tendons managed by early postoperative immobilization and tendons managed by early postoperative controlled passive motion.

Data analysis was based on the load / elongation uniaxial tensile test curve for each tendon. Load at Initial Failure (LIF), Stress at Initial Failure (SeIF), Strain to Initial Failure (SaIF), Material Stiffness (MS), Energy Absorbed to Initial Failure (EAIF) and cross-sectional area of the tendon callus, were all analyzed by a 2 x 5 (treatment condition x healing period) analysis of variance statistical procedure ($p \leq .05$).

Results included:

1. Significantly greater values for Load at Initial Failure (LIF), Stress at Initial Failure (SeIF) and Energy Absorbed to Initial Failure (EAIF) for the early controlled passive motion treatment group, throughout postoperative days 10 to 30 examined in this study.
2. For both treatment groups, across the 10 to 30 day healing period examined, there was a significant improvement in Material Stiffness (MS) and Stress at

Initial Failure (SeIF) and a significant decrease in Strain at Initial Failure (SaIF).

Planned orthogonal posthoc analysis further delineated significant changes in tensile property values for SaIF, MS and SeIF between days 20 and 25. Simple main effects posthoc analysis showed a significantly greater value for LIF, for the early controlled passive motion treatment group as compared to the immobilized treatment group, at the 25 day healing period.

Conclusions

Based on the results of this study, the following conclusions can be drawn:

1. A significantly greater Load at Initial Failure for the controlled passive motion treatment group as compared to the immobilized treatment group, was demonstrated throughout the 20 day healing period examined in this study. This finding suggests that flexor tendons managed clinically with early postoperative controlled passive motion will demonstrate a greater tolerance to tensile loads during the 10 to 30 day postoperative healing period. This finding of an increased rupture strength for early controlled passive motion tendons is consistent with other reported experimental studies.

2. This study has shown a significantly greater Stress at Initial Failure for the controlled passive motion treatment group as compared to the immobilized treatment group, throughout the 20 day healing period examined. This finding suggests that the controlled passive motion tendons increased tolerance to tensile loads may be due to actual morphological, or material differences between the two different treatment conditions examined in this study. However, any correlations drawn between morphological differences and differences in material strength are only speculative, as this study did not directly examine the morphology of the healing flexor tendons.
3. During the 10 to 30 day healing period examined in this study, both treatment groups demonstrated a significant increase in Stress to Initial Failure and Material Stiffness, and a significant decrease in Strain to Initial Failure. This finding is consistent with what would be expected to happen with these material tensile properties, if the healing tendon callus changes from a viscous material during the early portion of the healing period examined, to a more fibrous material during the later portion of the healing period.
4. This study further delineated significant changes in the material properties of Stress to Initial Failure,

Material Stiffness and Strain at Initial Failure, to between days 20 and 25. This time period correlates with reports in the literature of a histological change within the healing tendon from a process of fibroblast and collagen proliferation, to a process of collagen remodelling and maturation.

5. This study demonstrated a significantly greater Load at Initial Failure for the controlled passive motion treatment group as compared to the immobilized treatment group, at the 25 day healing period. As well, Energy Absorbed to Initial Failure increased for the controlled passive motion treatment groups and decreased for the immobilized treatment group, between the 20 to 25 day healing period. These findings suggest a possible morphological difference in the healing process during the early collagen remodelling and maturation phase of tendon healing, between the two treatment groups investigated.

6. This study was unable to demonstrate a significant difference in rate of change for any of the tensile properties examined, between the two treatment groups investigated in this study. This suggests that early controlled passive tendon mobilization regimes do not significantly alter the rate of healing in primarily repaired flexor tendons. Therefore, the assumption that early controlled passive motion tendons heal

more slowly than immobilized tendons was not substantiated by the findings of this study.

7. In a comparison of healing chicken FDP tendon tensile properties at the end of the thirty day healing period examined in this study, with normal chicken FDP tendon values for these same tensile properties determined in the pilot study, it was noted that there was a very large difference in magnitude between the values for healing and normal tendons for both treatment groups investigated in this study. This suggests that during the early stage of collagen maturation and remodelling that all healing flexor tendons should still be protected against excessive stresses and strains.

Clinical implications

Based on the experimental findings on a chicken animal model, possible clinical implications of this study are:

1. That the use of an early postoperative controlled passive motion treatment regime following primary flexor tendon repair may be an effective management regime for enhancing the overall rupture tolerance, and the actual material strength of healing flexor tendons in humans during the early postoperative healing period of 10 to 30 days.
2. That if it is considered clinically safe to institute

a protected active tendon mobilization program following three weeks of early postoperative immobilization, it should be equally safe to commence a protected active tendon mobilization program at this same time period following an early postoperative controlled passive motion regime.

3. It would be advisable during the early stages of active tendon mobilization for the clinician to proceed cautiously with graduation of the active tendon mobilization program, and to continue with some means of protective splinting to guard the healing tendon against excessive and unexpected strains.

REFERENCES

Amadio, P. C., & Hunter, J. M. (1987). Prognostic factors in flexor tendon surgery in zone 2. In: Tendon surgery in the hand (Hunter, Schneider & Mackin Eds.). Toronto: C. V. Mosby, pp 138-147.

Bartko, J. J., & Carpenter, W. T. (1976). On the methods and theory of reliability. J. Nervous and Mental Dis., 163(5): 307-317.

Bassett A. L. (1971). In: Physiological basis of rehabilitation medicine (Downey & Darling, Eds.). Toronto: W. B. Saunders.

Becker, H., Orak, F., & Duponselle, E. (1979). Early active motion following a beveled technique of flexor tendon repair: A report of fifty cases. J. Hand Surg., 4(5): 454-460.

Becker, H., Graham, M. F., Cohen, K., & Diegelman, R. F. (1981). Intrinsic tendon cell proliferation in tissue culture. J. Hand Surg., 6(6): 616-619.

Becker, H., & Diegelmann, R. F. (1984). The influence of tension on intrinsic tendon fibroplasia. Ortho. Rev., 13(3): 65-71.

Blanton, P. L., & Biggs, N. L. (1970). Ultimate tensile strength of fetal and adult human tendons. J. Biomech., 3: 181-189.

Booth, F. W., & Tipton, C. M. (1970). Ligamentous strength measurement in prepubescent and pubescent rats. Growth, 34: 1977-185.

Brokis, J. G. (1953). The blood supply of the flexor and extensor tendons of the fingers in man. J. Bone Jt. Surg., 35B: 131-138.

Bunnell, S. (1918). Repair of tendons in the fingers and description of two new instruments. Surg. Gyn. Obst., 26: 103.

Butler, D. L., Grood, E.S., & Noyes, F. (1978). Biomechanics of ligaments and tendons. Ex. Sport Sci. Rev., 6: 125-181.

Cannon, N. M., & Strickland, J. W. (1985). Therapy following flexor tendons surgery. Hand Clinics, 1(1): 147-165.

Duran, R. J., & Houser, R. G. (1975). Controlled passive motion following flexor tendon repair in zones 2 and 3. In AAOS: Symposiums on tendon surgury in the hand (pp 105-114). St. Loius: C. V. Mosby.

Duran, R. J., Houser, R. G., Coleman, C. R., & Stover, M. G. (1987). Management of flexor tendon lacerations in Zone 2 using controlled passive motion postoperatively. In: Tendon surgery in the hand, (Hunter, Schneider & Mackin. Eds.). Toronto: C. V. Mosby, pp 178-182.

Edwards, A. L. (Ed.). (1972). Experimental design in psychological research, 4th ed.. Toronto: Holt, Tinehart and Winston.

Edwards, D. A. W. (1946). The blood supply and lymphatic drainage of the tendon. J. Anat., 80: 147-152.

Ellis, D. G. (1969). Cross-sectional area measurements for tendon specimens: A comparison of several methods. J. Biomech., 2:175-186.

Elliott, D.H. (1965). Structure and function of mammalian tendon. Biol. Rev., 40: 392-421.

Elliott, D. H. (1967). The biomechanical properties of tendon in relation to muscular strength. Ann. Phys. Med., 9(1): 1-7.

Farkas, L. G., Thomson, H. G., & Martin, R. (1974). Some practical notes on the anatomy of the chicken toe for surgeon investigators. Plast. Reconstr. surg., 54(4): 452-458.

Farkas, L. G., Herbert, M. A., & James, J. S. (1980). Does early movement speed the recovery of function of repaired flexor tendon? Ann. Plast. Surg., 5(4): 305-308.

Forrest, L. (1983). Current concepts in soft connective tissue wound healing. Br. J. Surg., 54: 133-140.

Frank, C., Amiel, M.S., Woo, S. L-Y., & Akeson, W. (1984). Normal ligament properties and ligament healing. Clin. Orthop. Relat. Res., 196: 15-25.

Galante, J. O. (1967). Tensile properties of the human lumbar annulus fibrosus. Acta Ortho. Scand., suppl. 100.

Gelberman, R. H., Woo, S, L-Y., Lothringer, K., Akeson, W. H., & Amiel, D. (1982). Effects of early intermittent passive mobilization on healing canine flexor tendons. J. Hand Surg., 7(2): 170-175.

Gelberman, R. H., Manske, P.R., & Vandeberg, J.S. (1984). Flexor tendon healing in Vitro: Comparative histologic study of rabbit, chicken, dog & monkey. J. Orthop. Res., 2:39-48.

Gelberman, R. H., Vandeberg, J. S., Manske, P. R., & Akeson, W. H. (1985). The early stages of flexor tendons healing: A morphologic study of the first fourteen days. J. Hand Surg., 10A(6,part 1): 776-784.

Gelberman, R. H., Botte, M. J., Spiegelman, J. J. & Akeson, W. H. (1986). The excursion and deformation of repaired flexor tendons treated with protected early motion. J. Hand Surg., 11A(1): 106-110.

Goldin, B., Block, W. D., & Pearson, R. (1980). Wound healing of tendon. I: Physical, mechanical, and metabolic changes. J. Biomech., 13: 241-256.

Gordon, J. E. (1984). Structures: Or why things don't fall down. Markham: Penguin Books.

Graham, M., Becker, H., Cohen, K., Merritt, W., & Diegelman, R. F. (1984). Intrinsic tendon fibroplasia: Documentation by in vitro studies. J. Orthop. Res., 1: 251-256.

Green, W. L., & Niebauer, J. J. (1974). Results of primary and secondary flexor tendon repair in no-mans-land. J. Bone Jt. Surg., 56A: 1216.

Harkness, R. D. (1968). Mechanical properties of collagenous tissues. In Treatise on collagen (vol 2, part A, pp. 247-310). New York: Academy Press.

Hernandez, A., Velasco, F., Rivas, A., & Preciado, A. (1967). Preliminary report on early mobilization for the rehabilitation of flexor tendons. Plast. Reconstr. Surg., 40: 354-358.

Hester, T. R., Hill, L. & Nahai, F. (1984). Early mobilization of repaired flexor tendons within the digital sheath using an internal profundus splint: Experimental and clinical data. Ann. Plast. Surg., 12(2): 187-198.

Hirsh, G. (1975). Tensile properties during tendon healing. Acta Orthop. Scand., Suppl. 153.

Hitchcock, T. F., Light, M. D., Bunch, M. D., Knight, M. D., Sartori, M. J., Patwardhan, A. G., & Hollyfield, R. L. (1987). The effect of immediate constrained digital motion on the strength of flexor tendon repairs in chickens. J. Hand Surg., 12A(4): 590-595.

Hunter, J. M. (1987). The vincular system and its variations. In: Tendon surgery in the hand, (Hunter, Schneider & Mackin. Eds.). Toronto: C.V. Mosby, pp 24-45.

Kessler, I., & Nissim, F. (1969). Primary repair without immobilization of flexor tendon division within the digital sheath: An experimental and clinical study. Acta Orthop. Scand., 40: 587-601.

Ketchum, L. D. (1977). Primary tendon healing: A review. J. Hand Surg., 2(6): 428-435.

Ketchum, L. D., Mortin, N. L., & Kappel, D. A. (1977). Experimental evaluation of factors affecting the strength of tendon repairs. Plast. Reconstr. Surg., 59(5): 708-719.

Kleinert, H. E., Kutz, J. E., Ashbell, T., & Martinez, E. (1967). Primary repair of flexor tendons in "no-man's-land". J. Bone Jt. Surg., 49A: 577.

Kleinert, H. E., Kutz, J. E., & Cohen, M. (1975). Primary repair of zone 2 flexor tendon lacerations. In AAOS: Symposium on tendon surgery in the hand. St. Louis: C. V. Mosby. pp 91-104.

Kleinert, H. E., Schepels, S., & Gill, T. (1981). Flexor tendon injuries. Surg. Clin. North Am., 61: 267-286.

Kleinert, H. E., & Verdan, C. (1983). Report of the committee on tendon injuries. J. Hand Surg., 8(5, part2): 794-798.

Kleinert, H. E., & Cash, S. L. (1987). Current guidelines for flexor tendon repair within the fibro-osseous tunnel: Indications, timing and techniques. In: Tendon surgery in the hand, (Hunter, Schneider & Mackin. Eds.). Toronto: C.V. Mosby. pp 117-125.

Kutz, J. E. (1987). Controlled mobilization of acute flexor tendon injuries: Louisville technique. In: Tendon surgery in the hand, (Hunter, Schneider & Mackin. Eds.). Toronto: C. V. Mosby. pp 183-188.

Lahey, F. (1923). A tendon suture which permits immediate motion. Bost. Med. J., 188(22): 851-2.

Lindsay, W.K., & Thomson, H.G. (1959). Digital flexor tendons: An experimental study (Part 1). Significance of each compartment of the flexor mechanism in tendon healing. Br. J. Plast. Surg., 12: 289-316.

Lindsay, W.K., & Birch, J.R. (1964). The fibroblast in flexor tendon healing. Plast. Reconstr. Surg., 34: 223-232.

Lister, G. D., Kleinert, M. D., Kutz, J. E., & Atasoy, E. (1977). Primary flexor tendon repair followed by immediate controlled mobilization. J. Hand Surg., 2(6): 441-451.

Lister, G. D. (1985). Indications and techniques for repair of the flexor tendon sheath. Hand Clinics, 1(1): 85-95.

Lundborg, G. (1976). Experimental flexor tendon healing without adhesion formation. A new concept of tendon nutrition and intrinsic healing mechanisms. The Hand, 8(3): 235-238.

Lundborg, G., & Rank, F. (1978). Experimental intrinsic healing of flexor tendons based upon synovial fluid nutrition. J. Hand Surg., 3(1): 21-31.

Lundborg, G., Rank, F., & Heinau, B. (1985). Intrinsic tendon healing: A new experimental model. Scand. J. Plast. Surg., 19: 113-117.

Lundborg, G. N., & Rank, F. (1987). Tendon healing: Intrinsic mechanisms. In: Tendon surgery in the hand, (Hunter, Schneider & Mackin. Eds.). Toronto: C. V. Mosby. pp 54-60.

MacMillan, M., Sheppard, J. E., & Dell, P. C. (1987). An experimental flexor tendon repair in zone II that allows immediate postoperative mobilization. J. Hand Surg., 12A(4): 582-589.

Maddon, J. W., & Peacock, E. E. (1971). Studies on the biology of collagen during wound healing. III: Dynamic metabolism of scar collagen and remodeling of dermal wounds. Ann. Surg., 174(3): 511-518.

Manske, P. R., Bridwell, K., & Lesker, P. A. (1978). Nutrient pathways to flexor tendons of chickens using tritriated proline. J. Hand Surg., 3(4): 352-357.

Manske, P. R., & Lesker, P. A. (1983). Comparative nutrient pathways to the flexor profundus tendons in zone II of various experimental animals. J. Surg. Res., 34: 83-93.

Manske, P. R., Gelberman, R. H., Vandeburg, J. S., & Lesker, P. A. (1984). Intrinsic flexor tendon repair: A morphological study in vitro. J. Hand Surg., 66A(3): 385-396.

Manske, P. R., & Lesker, P. A. (1985). Flexor tendon nutrition. Hand Clin., 1(1): 13-24.

Mason, M. L., & Shearon, C. G. (1932). The process of tendon repair. An experimental study of tendon suture and tendon graft. Arch. Surg., 25: 615-692.

Mason, M. L., & Allen, H. S. (1941). The rate of healing of tendons. An experimental study of tensile strength. Ann. Surg., 113: 424-459.

Matsui, T., Jaeger, S. H., Merklin, R. J., & Hunter, J. M. (1987). Effects of injury to tendons and vincula on the vascular system of flexor tendons. In: Tendon surgery in the hand, (Hunter, Schneider, & Mackin. Eds.). Toronto: C. V. Mosby. pp 67-78.

Matthews, P., & Richards, H. (1976). Factors in the adherence of flexor tendon after repair. J. Bone Jt. Surg., 58B(2): 230-236.

Matthews, P. (1979). The pathology of flexor tendon repair. The Hand, 11(3): 233-242.

McDowell, C. L., & Snyder, D. M. (1977). Tendon healing: An experimental model in the dog. J. Hand Surg., 2(2): 122-126.

McGrouther, D. A., & Ahmed, M. R. (1981). Flexor tendon excursion in no-mans'-land. Hand, 13(2): 129-141.

Nelson, D. L., Heiple, K. G., Shaffer, J. W., Keith, M. W., Lacey, S. H., Davy, D. T., & Pazirandeh, P. H. (1985). The effects of intermittent passive motion on flexor tendon repair. Trans. Ortho. Res. Soc. 31st annual meeting. Los Vegas.

Nimni, M. B. (1983). Stucture, function and metabolism in normal and fibrotic tissue. Sem. Arth. Rheum., 13: 1-86.

Noyes, F. R., & Grood, E. S. (1976). The strength of the anterior curciate ligament in humans and rhesus monkeys. Age-related species-related changes. J. Bone J. Surg., 58A: 1074-1082.

Peacock, E. E. (1964). Fundamental aspects of wound healing related to the restoration of gliding function after tendon repair. Surg. Gynec. Obst., 119: 241.

Peacock, E. E., Madden, J. W., & Trier, W. C. (1971). Postoperative recovery of flexor tendon function. Am. J. Surg., 9A(1): 686-692.

Potenza, A. D. (1962). Tendon healing within the flexor digital sheath in the dog. J. Bone Jt. Surg., 44A(1): 49-64.

Ridge, M. D., & Wright, V. (1964). Description of skin stiffness. Biorheology, 2: 67-74.

Rosenblum, N. J. & Robinson, S. J. (1986). Advances in flexor and extensor tendon management. Clin. Phys. Ther., 9: 17-44.

Skoog, T., & Persson, B. H. (1954). An experimental study of the early healing of tendons. Plast. Reconstr. Surg., 13: 384-399.

Strickland, J. W., & Glogovoc, S.V. (1980). Digital function following flexor tendon repair in zone II: A comparison of immobilization and controlled passive motion techniques. J. Hand Surg., 5(6): 537-543.

Strickland, J. W. (1983). Management of acute flexor tendon injuries. Orth. Cl. North Am., 14(4): 827-849.

Strickland, J. W. (1985). Flexor tendon repair. Hand Clinics, 1(1): 55-68.

Tajima, T. (1984). History, current status, and aspects of hand surgery in Japan. Clin Orthop., 184: 41-56.

Thorngate, S., & Ferguson, D.J. (1958). Effect of tension on healing of aponeurotic wounds. Surg. 44: 619-627.

Tipton, C. M., Matthes, R. D., Maynard, J. A. & Carey, R. A. (1975). The influence of physical activity on ligaments and tendons. Med. Sci. Sport, 7: 165-175.

Urbaniak, J. R., Cahill, J. D., & Mortensen, R. A. (1975). Tendon suturing methods: Analysis of tensile strengths. In AAOS: Symposioum on tendon surgury in the hand. St. Louis: C. V. Mosby. pp 70-80.

Verdan, C. (1960). Primary repair of flexor tendons. J. Bone Jt. Surg., 42A: 647-657.

Verdan, C. (1972). Half a century of flexor tendon repair: Current status and changing philosiphies. J. Bone Jt. Surg., 54A: 472-491.

Viidik, A. (1973). Functional properties of collagenous tissues. Int. Rev. Conn. Tiss. Res. 2:127-215.

Vrijhoef M. M. A., & Driessens, F. C. M. (1971). On the interaction between specimen and testing machine in mechanical testing procedures. J. Biomech., 4: 233-238.

Wainwright, S. A., Biggs, W. D., Currey, J. D., & Gosline, J. M. (1976). Mechanical design in organisms. London: Edward Arnold.

Warren, C. G., Lehmann, J. F., & Koblanski, J. B. (1971). Elongation of rat tail tendon: Effect of load and temperature. Arch. Phys. Med & Rehab., 52: 465-474.

Weber, E. R. (1979). Synovial fliud nutrition of flexor tendons. Orthop. Res. Soc., 4:227-234.

Weber, E. R. (1987). Nutritional pathways for flexor tendons in the digital theca. In: Tendon surgery in the hand, (Hunter, Schneider, Mackin Eds.). Toronto: C. V. Mosby. pp 91-99.

Wray, R. C., & Weeks, P. M. (1980). Experimental comparison of technics of tendon repair. J. Hand Surg., 5(2): 144-148.

Yamada, H. (1970). Strength of biological materials (Evans, F. G., Ed.). Baltimore: Williams & Wilkins.

Young, R., Harmon, J. (1960). Repair of tendon injuries of the hand. Ann. Surg., 151: 562-566.

Young, V. L., Weeks, P. M., & Wray, R. C. (1981). Comparison of early passive motion and immobilization after flexor tendon repairs. J. Hand Surg., 6: 285.

APPENDIX I. Inter-rater reliability coefficients

A. Raw Data:

	LOAD (g)		STRAIN (%)		ENERGY (Joules)		AREA (mm^2)	
	R_1	R_2	R_1	R_2	R_1	R_2	R_1	R_2
1	1440	1500	10.00	10.25	.222	.212	2.84	2.82
2	825	840	5.00	5.00	.057	.055	2.66	2.60
3	1140	1150	3.00	3.00	.038	.038	2.31	2.25
4	1315	1325	5.50	5.50	.105	.091	7.94	7.74
5	1900	1900	5.00	5.00	.135	.123	2.66	2.72
6	540	575	6.00	5.75	.042	.041	-	-
7	825	820	4.50	4.50	.051	.046	-	-
8	1040	1025	5.00	5.00	.066	.066	-	-
9	1000	1000	6.00	5.25	.083	.084	-	-
10	660	675	5.50	5.00	.049	.046	-	-

B. Formulas:

1. One-way Anova

$$SS_B = \sum_{i=1}^{N} S_i^2/n_i - G^2/M \qquad M = N \times \#\text{ of raters}$$

$$SS_T = \sum\sum X_{ij}^2 - G^2/M \qquad df_B = N - 1$$

$$SS_W = SS_T - SS_B \qquad df_W = M - N$$

2. Inter-class Correlation Coefficient (ICC)

$$MS_B - MS_W \Big/ MS_B + (R - 1)\, MS_W$$

C. Calculations (ICC):

1. LOAD (LIF)

$SS_B = 293085.90$ $\quad MS_B = 325651.0$

$SS_W = 2862.00$ $\quad MS_W = 286.2$

$SS_T = 2933721.00$

ICC = .998

2. STRAIN (SaIF)

$SS_B = 59.215$ $\quad MS_B = 6.579$

$SS_W = 0.469$ $\quad MS_W = 0.047$

$SS_T = 59.684$

ICC = .986

3. ENERGY (EAIF)

$SS_B = 0.055020$ $\quad MS_B = 0.0061133$

$SS_W = 0.000239$ $\quad MS_W = 0.0000239$

$SS_T = 0.055259$

ICC = .992

4. AREA

$SS_B = 44.1346$ $\quad MS_B = 11.034$

$SS_W = 0.0256$ $\quad MS_W = .0032$

$SS_T = 44.1602$

ICC = .999

APPENDIX II. Raw Data

Raw Data Sheet - 1

Chicken #	H/P	X-sect (mm^2)		Load (grams)		Stress (g/mm^2)		Strain - a (t)	
		Immob	CPM	Immob	CPM	Immob	CPM	Immob	CPM
1	3	8.39	9.37	175	975	20.84	104.02	4.75	7.50
2	5	7.31	14.31	1775	2620	102.50	182.99	4.25	5.50
3	5	7.91	5.39	710	830	89.69	153.98	4.25	4.00
4	5	5.61	10.89	200	1175	35.61	107.83	4.00	4.50
5	1	6.87	11.70	500	740	72.68	63.24	9.00	7.00
6	2	6.49	14.11	290	750	44.68	53.12	7.25	5.75
7	1	12.03	9.48	250	920	20.77	97.02	4.00	12.50
8	2	10.89	11.19	975	820	89.44	73.28	5.25	4.75
9	4	8.91	8.94	675	1800	75.69	201.25	5.00	4.75
10	2	14.48	24.53	1150	2150	79.39	87.62	3.00	4.50
11	1	10.18	8.24	1075	450	105.60	54.59	5.75	5.00
12	4	5.11	16.50	775	2150	125.80	130.24	4.00	5.25
13	4	15.83	9.67	1525	2300	96.33	237.75	4.00	5.25
14	1	9.37	12.62	640	1360	68.28	107.70	3.50	8.00
15	4	12.08	13.68	1025	1325	93.23	96.80	5.25	5.75
16	3	6.64	20.42	420	1375	63.16	67.32	5.50	4.50
17	4	8.60	7.76	775	200	90.08	25.75	3.75	5.50
18	1	8.14	7.21	775	575	95.18	79.75	7.50	6.00
19	3	12.94	18.70	1450	1750	112.02	93.57	10.00	8.00
20	3	11.13	10.03	1090	1120	97.92	111.59	4.00	4.75
21	2	9.89	12.85	1090	1000	79.00	110.14	6.00	5.25
22	5	14.32	17.27	1900	1225	132.68	70.92	5.00	4.75
23	5	10.09	8.91	910	840	90.17	94.18	3.50	5.00
24	1	13.85	8.37	790	725	57.85	86.60	5.25	8.50
25	2	12.34	13.03	575	950	46.57	72.85	5.25	5.75
26	5	17.34	14.11	1450	2420	83.59	171.42	4.50	6.50
27	4	12.47	11.44	525	1475	42.10	128.38	5.25	3.25
28	3	10.12	12.37	850	550	83.99	44.44	8.00	5.50
29	2	14.08	8.76	1075	550	76.32	62.78	9.50	5.75
30	3	7.23	10.83	575	750	79.45	59.20	8.00	4.25

Raw Data Sheet - 2

Chicken #	H/P	Strain - b (%)		Stiffness-a (g/mm^2)		Stiffness-b (g/mm^2)		Energy (Joules)	
		Immob	CPM	Immob	CPM	Immob	CPM	Immob	CPM
1	3	4.00	6.25	438.76	1386.96	521.02	1664.35	.010	.112
2	5	3.50	5.00	2411.90	3327.90	2029.74	3859.98	.114	.197
3	5	3.75	3.00	2110.40	3849.60	2391.79	5132.80	.044	.043
4	5	3.75	3.75	890.48	2396.40	949.84	2875.68	.012	.068
5	1	8.00	6.00	807.60	903.54	908.55	1054.13	.072	.063
6	2	5.75	5.00	616.33	923.95	777.11	1062.54	.026	.069
7	1	3.25	11.25	519.28	77.82	639.11	86.24	.012	.146
8	2	4.75	3.75	1704.42	1542.74	1883.83	1954.13	.076	.051
9	4	4.25	4.25	1513.80	4236.88	1780.94	4735.34	.049	.115
10	2	2.50	4.00	2646.40	1947.18	3175.68	2190.57	.038	.141
11	1	4.75	4.00	1836.52	1091.84	2223.16	1364.80	.091	.029
12	4	3.25	4.75	3170.00	2480.76	3901.54	2741.89	.041	.172
13	4	3.25	4.25	2408.25	4528.59	2964.00	5594.14	.072	.169
14	1	3.00	7.25	1950.88	1346.33	2276.03	1485.60	.049	.163
15	4	4.50	4.75	1775.94	1683.60	2071.93	2038.04	.068	.105
16	3	4.50	4.00	1148.49	1496.07	1403.71	1683.08	.028	.089
17	4	3.25	5.50	2402.27	468.18	2771.85	468.18	.043	.023
18	1	6.25	5.00	1269.13	1329.17	1522.96	595.00	.085	.042
19	3	9.00	7.25	1120.21	1169.86	1244.68	1290.86	.222	.212
20	3	3.25	4.00	2448.12	2349.43	3002.25	2789.95	.057	.075
21	2	5.25	4.00	1316.68	2098.00	1504.78	2753.62	.091	.083
22	5	4.25	4.00	2653.62	1493.14	3121.91	1773.10	.135	.069
23	5	2.75	4.00	2576.29	2883.62	3278.91	2354.53	.039	.057
24	1	4.00	7.00	1101.98	1018.93	1446.35	1237.27	.058	.091
25	2	4.50	4.75	887.18	1267.09	1035.04	1533.85	.039	.085
26	5	4.00	6.00	1857.62	2637.32	2089.83	2857.10	.096	.262
27	4	4.75	2.75	673.62	3950.28	886.34	4668.51	.043	.066
28	3	7.00	4.50	1049.90	808.02	1199.89	987.58	.107	.041
29	2	8.25	4.75	803.45	1091.91	925.19	1321.79	.136	.039
30	3	7.00	3.25	993.16	1528.40	1135.04	2129.45	.083	.036